THE

# AMERICAN REVIEW OF TUBERCULOSIS

## ABSTRACTS OF TUBERCULOSIS

VOLUME XLIV NOVEMBER, 1941 ABST. No. 5

CONTENTS

Pages

Diseases Other than Tuberculosis

a. *Nontuberculous Infections of Lungs (concluded)* ..... 137–142
b. *Tumors of Lungs* ..... 142–147
c. *Miscellaneous Diseases of Lungs* ..... 148–161
d. *Extrapulmonary Conditions* ..... 161–164

**Atypical Bronchopneumonia.**—The usual textbook description of primary bronchopneumonia emphasizes the fact that this is essentially a disease of infants. In adults primary bronchopneumonia is stated to be rare, except when of the influenzal type as seen in pandemics, or of the streptococcal variety which is also epidemic in character as a rule. In the Presbyterian Hospital bronchopneumonia, as a primary disease of young adults, was usually considered a rare phenomenon; so rare, in fact, that a case presenting itself was usually suspected of having tuberculosis unless some other cause was apparent. Beginning with 1938, however, an increasing number of cases of primary bronchopneumonia was noted to which no specific aetiology could be ascribed, and which did not fall into any of the familiar classifications. The present article is a description of the clinical characteristics of this group and attention is focussed on it not only because of the number of cases and the severity of the disease, but also because the authors have the impression that they are dealing with a new disease entity. A fairly large number of instances are cited showing that the disease is a communicable one, and that the incubation period is quite long, being in the neighborhood of two or three weeks. The main clinical features are summarized as follows: The onset is more insidious than that seen in lobar pneumonia; the leucocyte count early in the disease is only slightly elevated, if at all; the sputum from a bacteriological standpoint is entirely comparable to that of perfectly healthy individuals; the pulmonary consolidation is incomplete and tends to be migratory; and the prognosis appears to be excellent. More than half of the cases presented themselves as comparatively mild, uncomplicated, bronchopneumonias conforming to this general pattern, with a febrile course of about ten days. Many cases, however, were much more severe, and in these certain features were moderately conspicuous, for example, the very prolonged course, the tendency to relapse, cyanosis with obstructed breathing and leucocytosis late in the disease. In a few cases occurring in females there was evidence of widespread affection of other systems, giving rise to the suspicion that there might be diffuse vascular lesions associated with the pneumonia. One sequence of cases is described which indicates quite strongly that the simple, the severe, and the complicated cases arose from the same aetiological agent. It is pointed out that the aetiology of the condition is obscure, and reasons are given against it being due to the various microorganisms recoverable from the sputum. Data relating to the possibility of a filterable virus being the causative agent are summarized but no proof of this hypothesis was found. One

fatal case is reported, together with the post-mortem findings. No final conclusions can be drawn from a single case, and the authors had no way of being sure that the individual in question was suffering from the same disease as the other patients in the series. This patient merely seemed to be, as far as they could tell, during the weeks under observation and treatment. Nevertheless it is extremely interesting that, histologically, the pneumonia proved to be of a most unusual variety, characterized by a mononuclear cell exudate and a tendency to organization. Furthermore, among other histological peculiarities, there was an associated acute vasculitis of the pulmonary vessels. One other feature of this case deserves comment. There also existed at autopsy an extensive purulent bronchitis with an exudate containing large numbers of type X pneumococcus. That this was an important contributing cause of death cannot be denied, and the abundance of this exudate suggested that it was partly responsible for the very extreme cyanosis at the end. The authors do not believe, however, that the type-X pneumococcus was related to the pneumonia. Sputum examinations early in the course of the illness failed to disclose this organism, and sulfapyridine therapy was without effect. Moreover, while the cells of the exudate in the bronchi were polymorphonuclear in type, those in the lungs were mononuclear, and almost no organisms were seen in the histological sections of the lung tissue itself. The authors are convinced that within the last two or three years a type of bronchopneumonia hitherto more or less unrecognized, and of undetermined aetiology, has become quite widespread, and that its clinical significance is sufficiently great to warrant this extended description. Case reports, temperature charts and reproductions of X-ray films and microphotographs of histological sections accompany the article.—*Current Bronchopneumonia of Unusual Character and Undetermined Etiology, Y. Kneeland, Jr. & H. F. Smetana, Bull. Johns Hopkins Hosp., October, 1940, 67: 229.*—(*J. S. W.*)

**Acute Respiratory Infection.**—A clinical and roentgenological study was made of an epidemic of acute respiratory tract infection occurring in Philadelphia from January to May 1939. The disease resembles influenza in many respects, but is caused by a different agent. It represents a clinical entity probably due to a filterable virus. The disease is primarily an inflammation of the mucous membrane of the respiratory tract, usually of the nose, pharynx and larynx, occasionally including the trachea and bronchi, and in a few instances the bronchioles and lungs. On the basis of this involvement the majority of the patients studied (87 per cent) were ill with nasopharyngolaryngitis, 7 per cent had in addition tracheobronchitis and 6 per cent had tracheobronchopneumonia. Three hundred seven patients (75 per cent of the cases) were sick but a few days. Twenty-five per cent of the cases were ill enough to go to bed; of this group, one-fourth had tracheobronchopneumonia; symptoms in this group consisted of generalized aching, pain in the eyeballs, photophobia, persistent hacking or paroxysmal cough, sore throat, anorexia; and occasionally nausea and vomiting; the temperature was usually elevated and averaged between 101°F. and 103°F., the fever lasting 2.5 to 4.6 days in nonpneumonia cases and 8.2 days in pneumonia cases; the leukocyte count ranged between 5,000 and 8,000 per cu. mm. of blood. Roentgenologically the disease manifested itself as either an acute tracheobronchitis (24 of 86 cases) or an atypical pneumonia (22 of 86 cases). The roentgen diagnosis of acute tracheobronchitis, admittedly very difficult, is based upon the finding of an increased prominence and blurred appearance of the hilum and trunk shadows and generalized haziness throughout the lungs. Two factors aided materially in arriving at a diagnosis of an acute tracheobronchitis. In the first place, the study was restricted to a select, young, healthy group of individuals, i.e. medical students, nurses and interns of the Jefferson Medical College and Hospital. Furthermore, the majority had had previous roentgen studies of the chest which were available for comparison. The pneumonic lesions showed great variability. One of the characteristic lesions consisted of a fairly well localized, but not sharply defined, area of increased density occurring predominantly in the lower lobes;

the areas were variable in size but never large; they varied considerably in density. Another fairly common type of lesion was a diffuse but not very dense pneumonic process at one base. In several cases the pneumonia showed a predilection for the hilar region, either unilateral or bilateral. At times the pneumonic reaction was indicated by a barely perceptible haziness, either unilateral or bilateral. The lesions usually cleared up in from a week to ten days, but in several cases the disease was protracted over a period of several weeks.—*The Roentgenological Aspects of an Epidemic of Acute Respiratory Tract Infection, K. Kornblum & H. A. Reimann, Am. J. Roentgenol., September, 1940, 44: 333.*—(*W. H. M.*)

**Sulfapyridine in Pneumonia.**—A review is given of the results of sulfapyridine treatment in 203 cases of childhood pneumonia. These are divided into 87 cases of bronchopneumonia, 46 males and 41 females, and 116 cases of lobar pneumonia in 59 males and 57 females. The average age in the former group was twenty-one months and in the lobar group four years and six weeks. The mortality was 2.5 per cent for the whole series. Several methods of administering the drug were used, namely, in tablet form to older children, in an emulsion for infants and occasionally in very sick children by intravenous drip using sodium sulfapyridine in normal saline. The dosages used varied but the average dose was 1.39 gr. per pound and the average total dose 158.74 gr. The author suggests, however, that a dosage of about 0.75 to 0.8 gr. per pound would be quite sufficient and would lessen the occurrence of toxic reactions. He also states that sodium sulfapyridine may be given intramuscularly, using 15 gr. in 20 cc. of sterile water. The average blood level of the drug after forty-eight hours' administration was 6.3 mg. per cent, which was higher than the 4 mg. per cent suggested by Long. The average duration of fever before chemotherapy was one hundred and thirty-six hours and after therapy thirty-nine hours, the fall in temperature usually being accompanied by a disappearance of the signs of toxaemia. Complications consisted of those caused by the infection and those considered as evidences of toxicity from sulfapyridine. In the former group there were 42 cases of otitis media, 5 mastoiditis, one lung abscess, 2 sinusitis and one empyema. Toxic drug effects were found in 74 cases and are listed as vomiting, diarrhoea, cyanosis, haematuria, skin eruptions, leukopenia and anaemia. When these occurred, administration of the drug was ceased. No toxic effects were noted in cases where the average dose was 0.88 gr. per pound. Examination of the urine should be made every two days for evidence of haematuria or sulfapyridine crystals. If such be found fluids should be forced and sodium bicarbonate given until the urine reaction is alkaline within twelve hours. —*Treatment of Pneumonia in Childhood, R. R. Struthers, Canad. M. A. J., March, 1941, 44: 271.*—(*W. G. C. M.*)

**Treatment of Pneumonia.**—In the period from June, 1939 to July, 1940, 190 adult patients with pneumococcal pneumonia were treated in the wards of the Johns Hopkins Hospital. The results obtained in the treatment of these patients have been compared with those of previous years. In 1935 to 1936 the mortality rate for pneumococcal pneumonia was 19.1 per cent and the mortality rate remained at this level until treatment by means of chemotherapeutic agents was available. In 1938 to 1939 the mortality rate was 7.2 per cent, and in 1939 to 1940 it was 7.9 per cent. In determining the effect of therapeutic agents in the treatment of pneumonia, certain factors must be taken into consideration. The age of the patient has a definite bearing since the prognosis in this disease becomes less favorable as the age of the adult patient increases. However, there has been no significant change in the age distribution of pneumonia patients during the years covered by this study. Another factor of considerable importance is the presence of a concurrent disease which might have an unfavorable influence on the course of pneumonia. Of the 190 patients with pneumococcal pneumonia which were treated in the most recent years, 36.3 per cent were suffering from one or more major concurrent diseases and this incidence was quite similar to that of the previous four years. Another important factor in assessing prognosis is whether bacteraemia is present.

During the years 1939 to 1940, 13.2 per cent of the patients had a pneumococcal bacteraemia at the time of admission to the hospital and this incidence is similar to that of previous years. The development of complications in the latest year of the survey is lower than in any other year. Three patients had empyema on admission to the hospital and 2 developed empyema after specific therapy had been instituted. All of the empyemas were caused by Type-I pneumococci. Of the 190 patients 10 developed sterile pleural effusions while in the hospital. In planning treatment of pneumococcal pneumonia in adults it was decided that specific antipneumococcal serum would be used only in those patients who appeared to be very ill on their admission to the hospital. In all other patients sulfapyridine or sulfathiazole was used. A routine peroral dosage schedule for either drug was followed. An initial dose of 4.0 g. was given as soon as the clinical diagnosis of pneumonia had been established. Then 1.0 g. was given every four hours day and night until the temperature had been normal for forty-eight hours. This was followed by 1.0 g. every six hours until resolution of the pneumonia was well advanced; and finally 0.5 g. was given four times a day until the patient's lungs were free from signs of pneumonia. If on the day after the beginning of treatment the rectal temperature of the patient had not dropped to below 101°F. and the concentration of free sulfapyridine or sulfathiazole in the patient's blood was under 4 mg. per cent, then one dose of 0.06 g. per kilogram of body weight of either sodium sulfapyridine or sodium sulfathiazole was given intravenously. Twenty-five of the 190 patients received type-specific serum and sulfapyridine; 12 received serum and sulfathiazole; 5 were given serum and both drugs; 68 were treated with sulfapyridine alone; 66 with only sulfathiazole; and 13 received both sulfapyridine and sulfathiazole. In most instances the use of antipneumococcal serum in patients with Type-I pneumococcal pneumonia was on an experimental basis for another study. However, in this group of 190 patients, 20, or approximately 10 per cent, were thought to be so ill as to actually require intensive serum and drug therapy. In the entire group of 190 patients 15 deaths occurred, a fatality rate of 7.9 per cent. In 6 instances death resulted primarily from a major concurrent disease. In 7 cases the patient died within twelve hours after treatment had been instituted. In only 2 patients did death occur despite apparently adequate treatment. The need for early intensive specific treatment of pneumonia cannot be too strongly emphasized. Sulfathiazole is absorbed more rapidly and excreted more rapidly than is sulfapyridine. The former drug is found to be distributed fairly evenly in the tissues and seems to pass readily into infected pleural fluids. Because of the rapid excretion of the drug it must be administered every four hours in order to maintain adequate concentration in the blood. Therapeutically effective concentrations of sulfathiazole are between 4 and 6 mg. per cent. Drug rashes and drug fevers occurred more frequently in those patients treated with sulfathiazole than with those who received sulfapyridine; however, sulfathiazole caused less nausea and vomiting than did sulfapyridine. It is felt that sulfathiazole and sulfapyridine are equally effective in the treatment of pneumococcal pneumonia in adult patients. Patients who are severely ill should receive both type-specific serum and either sulfathiazole or sulfapyridine.—*The Problem of Pneumonia with Reference to Chemo- and Sero-Therapy, P. H. Long & J. W. Haviland, Ann. Int. Med., December, 1940, 14: 1042.*—(*A. A. E.*)

**Bronchography in Suppurative and Neoplastic Disease.**—Before deciding upon a major surgical procedure as a treatment for bronchiectasis, it is essential to visualize every bronchial division by means of bronchography. Thick secretions may partially block a bronchus and prevent filling of bronchiectatic areas. Repetition of bronchography after postural drainage or introduction of iodized oil by the bronchoscopic route should be attempted. It is advisable to attempt filling of only one side of the bronchial tree on any one day. The right anterior oblique view will best visualize the branches of the left main bronchus, and the divisions of the right bronchial tree are most clearly separated in the left anterior oblique view. In some cases roentgenograms in frontal and lateral projections are also desirable. In

57 per cent of cases with left lower bronchiectasis the writers found that the lingula of the left upper lobe was also diseased. Right middle lobe involvement was demonstrated in 59 per cent of right lower lobe bronchiectasis. Nontuberculous abscesses of the lung can rarely be filled with iodized oil given by the supraglottic method. Bronchoscopy with aspiration of secretion preceding instillation of the oil is more often successful in demonstrating the cavity. If the bronchus leading to the abscess is blocked, the position of the lesion and its relationship to the chest wall can be deduced by determining the identity of the obstructed bronchial division. Although bronchography gives important information concerning the location of bronchogenic carcinomas situated in the major bronchial divisions, bronchoscopy is more accurate.—*The Value of Proper Interpretation of Lipiodol Bronchograms in Thoracic Surgery, I. W. Myers & B. Blades, Am. J. Roentgenol., October, 1940, 44: 530.*—(*W. H. M.*)

**Asthmatic Bronchitis in Children.**—The author presents a study of 235 consecutive cases of so-called asthmatic bronchitis in children, and attempts to define a clinical entity as distinguished from asthmatic bronchitis of allergic origin and to prove that this condition is frequently found in chronic infections of the upper respiratory tract. The symptom complex includes frequent attacks of bronchitis accompanied by wheezy respiration during the first few years of life, recurring colds, anorexia, abdominal pain and nausea, night sweats and malodorous morning breath. The laboratory reveals severe secondary anaemia, often as low as 45 per cent, a slightly increased leucocytosis and relative neutrophilia, and usually pure nasopharyngeal cultures of streptococci or staphylococci, or a mixed growth. These attacks differ from asthma in being continuous rather than paroxysmal, they do not respond satisfactorily to epinephrine, allergy plays little part, local infection is constantly present and eosinophilia absent. In the author's treatment experience, removal of tonsils and adenoids in 30 per cent of the cases had no apparent influence. Diet, codliver oil, rest, treatment of the anaemia, local epinephrine and specific autogenous vaccine therapy seem to afford the best results. Sulphanilamide is useful in asthmatic bronchitis but valueless in the chronic infection. A negative tuberculin test is important, since there is often suspicion of tuberculosis, and the roentgenologist is frequently led astray by shifting areas of pulmonary bronchopneumonic infiltration and a characteristic peribronchial reaction which often results in a roentgenographic diagnosis of primary infection tuberculosis. — *Asthmatic Bronchitis following Chronic Upper Respiratory Infection: A Five Year Study of 235 Consecutive Cases Seen in Private Practice, L. Bivings, J. A. M. A., October 26, 1940, 115: 1434.*—(*G. L. L.*)

**Pulmonary Complications of Renal Suppuration.**—One hundred and seventy-nine cases of pyonephrosis (8 tuberculous) and 85 cases of perinephric abscess (5 tuberculous) were collected at the University Hospital (Michigan) over the thirteen-year period from 1925 to 1938. Only 3 (1.7 per cent) of the patients having pyonephrosis showed supraphrenic complications, whereas there were 14 such cases (16.5 per cent) in the group of patients with perinephric abscess. Most of the patients showed pleuritic changes only, but 4 probably had pneumonia of some degree and 6 had perinephro-bronchial fistulae. In all but one patient the changes were evident on roentgen examination of the chest. All patients showed elevation of the diaphragm on the affected side. In some cases the renal condition was overshadowed by the pulmonary complication and surgical treatment of the renal or perirenal suppuration was delayed. Except in the presence of a frank empyema or pulmonary abscess the treatment of the pulmonary complications consists of an adequate drainage of the primary lesion.—*Pulmonary Complications of Acute Renal and Perirenal Suppuration, R. M. Nesbit & V. S. Dick, Am. J. Roentgenol., August, 1940, 44: 161.*—(*W. H. M.*)

**Exudates of Subphrenic Origin.**—Pleural exudates may occur as sympathetic effusions due to adjacent subphrenic abscess or there may be actual communication through the diaphragm between a subphrenic abscess and

an empyema cavity; occasionally the subphrenic abscess may extend through the diaphragm, burrow along the interlobar fissure and rupture into a bronchus. Malignant infiltration and erosion through the intestinal wall, perforating lesions of the stomach, duodenum or appendix, and perinephric abscess may serve as aetiological factors in subphrenic involvement. Occasionally subphrenic abscess follows an abdominal operative procedure. Ordinary methods of roentgenological examination often fail to disclose subphrenic lesions as a basis for conditions found in the thoracic cavity. Artificial pneumoperitoneum may be resorted to as a diagnostic agent without fear or breaking down of adhesions or spread of infection. By this method air enters freely into the subphrenic region on the uninvolved side but is prevented from entering on the involved side by adhesions resulting from subphrenic abscess.—*Basal Exudates of Subphrenic Origin, L. R. Sante, Am. J. Roentgenol., September, 1940, 44: 350.*—(*W. H. M.*)

**Cold Vaccines.**—The authors follow up a report made more than a year ago of a series of controlled studies of several vaccines, including a mixed bacterial vaccine in which the organisms were destroyed mechanically rather than by heat, with a further report of the value of the traditional type of heat-killed bacterial vaccine for the prevention of the common cold. Experimental and control groups comparable with those of the previous report were set up, and 77.3 per cent of the vaccinated group of 119 students and 83 per cent of the control group of 106 cold-susceptible students at the University of Minnesota were followed to the end of the experimental period. The vaccine used consisted of the following heat-killed organisms per cubic centimeter: *Staphylococcus aureus* and *Staphylococcus albus*, of each one billion; streptococcus, pneumococcus, *Micrococcus catarrhalis*, Friedländer's bacillus and influenza bacillus, of each 400 million. During the year, while the vaccinated students reported an average of 2.1 colds per person which represented a reduction of 55 per cent as compared with an average of 4.7 colds for the previous year, it was noted that the control group reported a reduced average of colds representing a decrease of 61 per cent as compared with the year previous to the study. It is apparent therefore that treatment with the traditional heat-killed bacterial vaccine has little or no influence on the average incidence of colds. It was noted that many of the students receiving the vaccine had quite severe reactions. —*Cold Vaccines: A Further Evaluation, H. S. Diehl, A. B. Baker & D. W. Cowan, J. A. M. A., August 24, 1940, 115: 593.*—(*G. L. L.*)

**Influenza and Lung Tumors in Mice.**—A report is given of an experimental study of the influence of the influenza virus infection on the incidence of primary lung tumors in mice. After an extensive experience with the pathological aspects of influenza during the 1918 pandemic, Winternitz and his colleagues prophesied an increase in the incidence of lung carcinoma because of the metaplasia of the bronchial epithelium and proliferation of young cells produced in the lungs. The combined effects of influenza virus and exposure to an atmosphere containing carcinogenic dust from tarred roads were studied in mice over a period of three years. Each mouse was initially infected with influenza virus and subsequently exposed to this dust for a period of a year. Of 23 mice so treated only 13 lived to the age of ten months or longer. There were 2 cases of lung tumors in these 13 mice, or an incidence of 15.4 per cent. Both tumors were malignant and their hosts were 605 and 877 days old respectively. In a control group of similar age the incidence of lung tumors in dusted mice was 31.5 per cent and in not specially dusted mice 11.3 per cent. It appears that the influenza virus inhibited the development of primary lung tumors in mice in this small series.—*Influenza Virus and the Incidence of Primary Lung Tumors in Mice, J. A. Campbell, Lancet, October 19, 1940, 2: 487.*—(*J. B.*)

**Malignant Tumors of Lung.**—A biopsy demonstrating the presence of a malignant tumor should be obtained if possible before radical treatment is instituted. This is sometimes difficult in cancer of the lung because the tumor may be extrabronchial or it may be well out in the lung field beyond the accessibility for bronchoscopic biopsy. In 175 cases of

proven cases of cancer of the lung seen at Memorial Hospital, the material for histological diagnosis was made by lung puncture in 64 cases. Punch biopsy is always done with the patient in the prone position since fear of air embolism has led to the abandonment of the performance of the aspiration with the patient sitting up. In small tumors the aspiration procedure may be done under fluoroscopic guidance. Under novocaine anaesthesia the eighteen gauge needle on a syringe is inserted through the predetermined point into the tumor. When one is sure they are in the tumor, the plunger of the syringe is pulled out firmly and steadily so suction is applied. When the needle is removed the tissue which is in the needle is pushed out onto a slide with a stylet. In a series of 230 lung punctures for biopsy there were 2 cases of air embolism. Both of them occurred before the prone position was used. The patient may occasionally expectorate some blood. In this series there was no empyema or lung abscess. Occasionally it is also possible to find tumor cells in the sputum. —*Diagnosis of Malignant Lung Tumors by Aspiration Biopsy and by Sputum Examination, L. F. Craver, Surgery, December, 1940, 8: 947.*—(*H. W. H.*)

**Bronchogenic Carcinoma.**—For some unknown reason there has been a marked increase in the incidence of carcinoma of the lung. At Charity Hospital in New Orleans there were 7,433 autopsies on individuals over one year of age between January 1, 1931 and June 30, 1939. Among these there were 92 cases of carcinoma of the lung, 169 cases of carcinoma of the stomach and 56 cases of carcinoma of the biliary system. In other words carcinoma of the lung was more than one half as common as cancer of the stomach and almost as frequent as carcinoma of the pancreas and the biliary system together. Carcinoma of the lung was more common in the whites than the colored. It was much more common in males than females. The lesion was slightly more common on the right side. The gross appearance yields no criteria by which microscopical structure of the carcinoma can be predicted. The cells of these tumors seem to arise from the reserve cells, the undifferentiated cells lined up along the tunica propria. The growths are classified as squamous-cell, columnar-cell, and reserve-cell carcinomata. These occurred in the proportion of 5:2:3. Distant metastatic foci were found in 81 of the 92 cases.—*Morphologic Aspects of Carcinoma of the Lung, B. Halpert, Surgery, December, 1940, 8: 903.*—(*H. W. H.*)

**Bronchogenic Carcinoma.**—The authors give a review of the present status of cancer of the lung. The incidence has increased both relatively and absolutely. Autopsy statistics place it second only to cancer of the stomach. The increase is believed to be due, in part, to the increase in cigarette smoking. It is not settled which of the constituents of tobacco is responsible. The association of cancer of the lung with the inhalation of radioactive substances was established in the case of miners in Schneeberg mines. Approximately 80 per cent of the cases occur in males. The highest incidence is reached in the sixth decade. About 85 per cent of the cases are above forty and 65 per cent are above fifty. The disease affects the right lung slightly more frequently than the left. The authors believe all pulmonary carcinomata to be of bronchogenic origin, and they have followed Halpert's classification into three groups. The most primitive type is formed by the primitive endoderm cell of the bronchial mucosa, which they call the "reserve" cell. The next group consists of cuboidal cells, and the third group is the typical squamous cell carcinoma. Metastasis occurs by extension, intrabronchial spread and lymphatic and haematogenous spread. The regional lymph nodes are involved in 75 per cent of the collected cases. Other frequent sites are the liver, bone, adrenal, kidney, brain, heart and pericardium and pancreas. There is a paucity of early symptoms, and the disease should be suspected in a patient over forty complaining of cough, haemoptysis, or thoracic discomfort of any kind. The diagnostic means include X-ray examination, bronchoscopy, bronchography, examination of the sputum for malignant cells and biopsy. The authors condemn aspiration biopsy because of the danger of pleural metastasis, and advise bronchoscopic biopsy whenever possible. The treatment of choice is total pneumonectomy which permits a radical

dissection of the mediastinal glands. Recovery after pneumonectomy is around 45 per cent.—*Carcinoma of the Lung, A. Ochsner & M. DeBakey, Arch. Surg., February, 1941, 42: 209.*—(*H. M.*)

**Bronchogenic Carcinoma.**—Variations in type of bronchopulmonary cancer are reviewed and a histological classification is presented. Forty-six figures illustrating the gross and microscopical pathology of different varieties of lung tumor are included. The author recommends embedding sputum in paraffin and sectioning as an aid to diagnosis. In 80 to 85 per cent of cases of pulmonary carcinoma characteristic neoplastic cells can be recognized. Similarly examination of the sediment of pleural fluid permits recognition of tumor in about 60 per cent of cases. Lung puncture enables diagnosis in 60 per cent and bronchoscopic examination in 75 per cent of cases.—*Diagnostico anátomo-patológico del cáncer bronco-pulmonar, D. Vivoli, An. Catedra de pat. y clin. de tuberc., June, 1940, 2: 32.*—(*E. R. L.*)

**Bronchogenic Carcinoma.**—One hundred and seventy-five cases of bronchogenic carcinoma proved by histological study were collected at the Memorial Hospital from 1918 to 1939. The average age of the patient at the time of onset was fifty-three years and 90.3 per cent were males, 9.7 per cent females. Thirty-three per cent, or 58 cases, were accepted as proved cases of primary bronchogenic carcinoma on the basis of aspiration biopsy. Removal of a biopsy by bronchoscopy is more likely to show cellular arrangement and thus to permit a diagnosis of type, grade and radiosensitivity of the tumor more readily than the usual aspiration biopsy. However, bronchoscopy may not reveal tumor tissue, and aspiration biopsy is then performed. Forty-nine per cent of the cases were classified as epidermoid carcinoma, 10 per cent as adenocarcinoma, and 11 per cent as anaplastic carcinoma; in addition 9 per cent were classified simply as bronchogenic carcinoma and 21 per cent as "carcinoma", often the best diagnosis that could be made from the bits of tissue obtained by bronchoscopy or aspiration biopsy. The study of results of treatment in the 175 cases is based on a follow-up which shows 20 patients living, 140 dead and 15 untraced. Seventeen patients were untreated. The total duration of the disease in 14 of the latter, from the time of apparent onset until death, averaged 14.8 months, and varied from 2.2 to 39 months. Review of cases treated by roentgen rays showed that as the tumor dose in roentgens is increased in the initial cycle (many patients had several cycles of therapy) the duration of life following treatment increases. Cases receiving from 2,000 r to 3,000 r in the tumor survived an average of 8.2 months, as compared with an average survival of only 4.3 months in those receiving less than 1,000 r in the tumor. In the 3,000 to 4,000 r group, the average survival was 5.4 months, but there were only 4 cases in this group. Considering either total doses or doses in the first cycles only, there is a marked trend toward improved palliation as the tumor dose increases. As between the various histological types of carcinoma, there seems to be hardly a significant difference in the survival figures, but the grades of palliation seem to favor the epidermoid group as against the others. Based on all proved cases regardless of whether treated or untreated, followed or lost, the five-year survival rate is only 2.8 per cent. Based on cases treated by roentgen rays it is 3.8 per cent. Even the two-year survival rate in cases treated by roentgen rays is only a little under 7 per cent. Therefore, we still have to point largely toward palliative effect and possible prolongation of life in speaking of the value of irradiation in carcinoma of the lung. An increase in the tumor dose with less irradiation of surrounding tissue will be accomplished in the future by careful cross-firing, long target-skin distances, higher voltages, greater filtration, and restriction of the portals to a field large enough to include thoroughly only the estimated extent of the tumor, ignoring peripheral shadows in the roentgenograms thought to be caused by secondary atelectasis and inflammatory changes. Of the 20 living patients, one is apparently free of disease ten months after lobectomy. The remaining 19 were all treated by roentgen rays. One of these, also treated with radium, living and well 12.5 years, had

considerable necrosis of the chest wall in the tenth year, necessitating surgery; a further complicating empyema cleared up. Two patients have almost attained the five-year survival. A "two year" patient and an "eighteen months" patient are seriously ill. Of 13 patients alive six months or less, several are preterminal. However, all but one patient have had some degree of palliation.—*Bronchiogenic Carcinoma, L. F. Craver, Am. J. Roentgenol., April, 1940, 43: 469.*—(*W. H. M.*)

**Bronchogenic Carcinoma.**—Two cases of proved primary carcinoma of the lung one of which lived more than seven months after having been given deep roentgen-ray therapy; the other, an advanced case with metastasis, died eighteen days after admission to the hospital. The diagnosis in both cases was confirmed by bronchoscopy and biopsy. One case illustrated a fairly good palliative result after deep roentgen-ray therapy, in which the tumor definitely regressed. In the other case the primary tumor was quite small. The metastasis to the liver was extensive, the latter being the main disturbing factor to the patient. The roentgen picture of primary carcinoma of the lung, although striking, is not diagnostic. It must be interpreted in the light of the history and clinical findings. Bronchoscopy and biopsy are necessary in order to confirm the diagnosis and to determine the cell type while the patient is living.—*Primary Carcinoma of the Lung, C. H. Kelly, Radiology, July, 1940, 35: 61.*—(*G. F. M.*)

**Bronchogenic Carcinoma.**—A review of the literature on carcinoma of the lung makes it evident that a definite diagnosis cannot be made without biopsy specimen and microscopical studies. These studies have, however, produced information which greatly aids in a proper evaluation of important signs and symptoms as well as methods of differentiation from other pulmonary diseases. The majority of cases occur between the fortieth and seventieth years. Carcinoma of the lung is much more common in males than females. The lesion is found more commonly on the right side. Early symptoms vary widely but cough and chest pain are important early symptoms. Dyspnoea may be distressing but is usually a late symptom as are weight loss, cachexia, nightsweats, fever, Horner's syndrome and clubbing of the fingers. It is quite generally accepted that all carcinoma of the lung arises from the basal cells of the bronchial mucosa. In some cases diagnosis is relatively simple and in others it is necessary to make use of the roentgenogram, fluoroscope, bronchoscope and thoracoscope. Biopsy specimens may at times be obtained through either of the last mentioned instruments. If there is pleural fluid present this may be aspirated and the cell content studied. In a certain group of these cases the definite diagnosis is made only by biopsy of metastatic lesions.—*Primary Bronchiogenic Carcinoma, J. J. Singer, Surgery, December, 1940, 8: 910.*—(*H. W. H.*)

**Bronchogenic Carcinoma.**—The bronchoscopic diagnosis of bronchogenic carcinoma is based upon the interpretation of pathology revealed by direct inspection of the bronchial tree. By such an examination it is possible to study the character of the lesion, to observe its location, to obtain tissue for biopsy and to aid in determining operability. The most frequent symptoms of an intrabronchial new growth are cough, haemoptysis and wheeze. The presence of these symptoms is an indication for bronchoscopic examination, as are roentgen findings which suggest bronchial obstruction. Since over 80 per cent of the neoplasms arise in the major bronchi they will not produce obstructive signs early, therefore one should not wait until these signs appear, but a bronchoscopic examination should be done in the presence of unexplained cough, haemoptysis or wheeze. Tumors which narrow the bronchial lumen, whether they are intrabronchial, peribronchial or extrabronchial produce the signs of obstruction, that is wheeze, atelectasis or obstructive emphysema. Biopsy material can be obtained through the bronchoscope in approximately 75 per cent of cases. The bronchoscope is also useful in determining the site, and therefore the operability of the lesion. If all other symptoms and signs point to carcinoma and no positive biopsy is obtainable, this fact should not contraindicate surgery, because in a certain group the tumor is located

at a point too distal to be observed through the bronchoscope. With the exception of a few benign bronchial tumors, bronchoscopic therapy is not successful except as palliative measures.—*Bronchoscopic Diagnosis of Bronchial Carcinoma, P. Holinger & D. B. Radner, Surgery, December, 1940, 8: 939.*—(*H. W. H.*)

**Bronchogenic Carcinoma.**—The most nearly pathognomonic of the roentgenological changes indicative of primary carcinoma of the lung is a unilateral increase in density in the hilum. Of almost equal importance is some degree of atelectasis. One or both of these changes were observed in about two-thirds of 206 proved cases which were seen at the Mayo Clinic from 1928 to 1937. The presence of these signs should suggest at once the possible existence of bronchial malignancy. Likewise, the presence of an ill-defined or rounded shadow of increased density, away from the region of the hilum, should be considered indicative of malignancy until proved otherwise. Not infrequently, the earliest roentgenological changes associated with bronchogenic carcinoma are those which may readily be confused with a benign inflammatory process. In a few such instances, the demonstration of displacement phenomena with the presence of abscess or bronchiectasis, the coexistence of atelectasis and hydrothorax, or the recognition of an elevated and immobile diaphragm on one side, may suggest the presence of malignancy. More often, however, the diagnosis of carcinoma will be considered only if its possibility is kept in mind. A definite diagnosis or at least a suggestion of its presence was made in about 60 per cent of the 206 cases. In the remaining 40 per cent, the condition in one-third was mistaken for inflammatory lesions of the thorax; in one-third a merely descriptive report was made, and in the remainder the lesion was variously diagnosed as lymphoblastoma, metastatic carcinoma, interlobar fluid, tuberculosis, aneurysm, or "negative chest."—*Primary Carcinoma of the Lung, J. W. Olds & B. R. Kicklin, Am. J. Roentgenol., September, 1940, 44: 357.*—(*W. H. M.*)

**Excavated Bronchogenic Carcinoma.**—Characteristic features of primary carcinoma of the bronchus are sufficiently well known today so that with the use of modern methods the diagnosis is, in general, established readily. In many instances, however, the findings so closely simulate chronic pulmonary infection that the possibility of cancer may be overlooked. In a series of 127 cases of primary bronchogenic carcinoma admitted to Cleveland City Hospital from 1927 to 1937 proved by autopsy, bronchoscopic or operative biopsy and in which roentgenological examination was performed, 12 per cent had roentgenological evidence of cavity formation which necessitated the differentiation from tuberculosis and lung abscess. The important symptoms of these 15 patients were those common also to tuberculosis and lung abscess and their histories did not reveal any clinical picture which could be considered pathognomonic of a cavitary bronchogenic carcinoma. The most important finding consisted of decreased breath sounds and voice sounds in the involved area, indicating the presence of some degree of bronchial obstruction, which may also occur in abscess and tuberculosis. Of the 15 patients, a correct diagnosis of carcinoma of the bronchus was made in 10; 6 were established by bronchoscopic examination and by biopsy of the lung at thoracotomy in 4 others. In 4 of the cases erroneously diagnosed, study of the autopsy specimens indicated that bronchoscopy would probably have revealed the correct diagnosis. From these findings it has become the rule of the Cleveland City Hospital that all cavitary lesions of the lung which cannot be definitely established as tuberculous in origin should be examined bronchoscopically to aid in excluding the presence of an underlying carcinoma, especially in males above the age of forty years, with the clinical picture of chronic lung abscess.—*Cavitary Bronchogenic Carcinoma, H. Hauser & S. E. Wolpaw, Radiology, June, 1940, 34: 698.*—(*G. F. M.*)

**Bronchogenic Carcinoma of Lung in Chinese.**—Up to the present time there are very few reports on primary carcinoma of the lung in the Chinese. However, this disease is not very rare there, though not so frequent as in the Western countries. The incidence has increased in recent years, probably due to

better diagnostic methods. In this paper 21 cases are reported which were observed in the Peiping Union Medical College Hospital during the past twenty years. The diagnosis was confirmed in 3 cases by postmortem examination, in 5 cases by biopsy through thoracotomy or aspiration of the lung and in 10 cases after biopsy of the regional lymph nodes, subcutaneous nodule or bone metastasis or study of the sputum or aspirated pleural fluid. The age of the patients was thirty-five to sixty-nine years. There were 15 male and 6 female patients. The symptoms were similar to those described in text-books. Only in 3 cases no metastasis could be found. The value of the roentgenological examination for the diagnosis is stressed; the roentgenological findings in this disease are extensively described. According to the location the central and the peripheral types are distinguished, the first being divided into the stenosing and the nonstenosing forms. The X-ray pictures are reproduced and discussed.—*Primary Carcinoma of the Lung in the Chinese, C. K. Hsieh, S. H. Wang & F. C. Chang, Chinese M. J., October, 1940, 58: 381.*—(*G. C. L.*)

**Bronchographic Examination of Bronchogenic Carcinoma.**—By serial bronchography under roentgenographic control it is possible to localize bronchogenic growths, determine their extent, and to differentiate polypoid, infiltrating and mixed types. Uroselectan is preferred to iodized oil because it is less viscid and is rapidly absorbed. The polypoid types of tumor are most frequently found in the major bronchi near the carina but may arise at the entrance of a lobar bronchus. They produce round or irregular filling defects in the bronchial lumen. Below the filling defect the bronchus is always dilated, and in this portion round and irregular filling defects are caused by retained bronchial secretion. The infiltrating forms are characterized by invasion of the mucosa or submucosa of the bronchus to varying degrees. In the main or lobar bronchi, incomplete infiltration produces irregularity and concentric narrowing of the lumen; when the process is advanced, producing bronchial occlusion, the filled lumen has the appearance of an opaque cone. When the neoplastic infiltration is situated in bronchi of the third or fourth order, complete obstructions predominate because the lumen in these bronchi is small; concentric narrowing may be observed early. In bronchi of very small calibre, the lumen is usually obstructed very rapidly, and the usual picture is that of a peripheral nodular growth compressing and displacing the adjacent bronchi filled with contrast substance. The mixed forms exhibit the findings of the polypoid and infiltrating lesions. They are usually found in the major or lobar bronchi, less often in bronchi of the third or fourth order.—*Recent Progress in the Bronchographic Examination of Bronchogenic Carcinoma, P. L. Farinas, Am. J. Roentgenol., September, 1940, 44: 370.*—(*W. H. M.*)

**Tomography in Tumors of Lung.**—Cancer of the lung comprises 5 to 10 per cent of all cancers. With the recent advances in thoracic surgery removal of the cancer by lobectomy or pneumonectomy has been accomplished with brilliant results. Mortality from these operations is being greatly reduced. It is estimated that 65 per cent of bronchogenic carcinomata can be diagnosed by bronchoscopy. Body-section radiography should reduce the remaining 35 per cent. Body-section radiography is a general term covering the various methods of localizing the tumor at a definite level by making the X-ray exposure while the tube and the film are moving. Body-section radiography is of greatest use anatomically where there is a maximum superimposition of structures. It is especially valuable in the examination of the respiratory tract; and this is particularly true in the upper portion of the lungs, which are difficult to examine with the bronchoscope. With growing experience with this method of examination, far earlier diagnosis of malignant tumor of the lower respiratory tract can be made to the consequent benefits of these unfortunate patients.—*Body-Section Radiography in Malignancy of the Lower Respiratory Tract, S. Moore, Surgery, December, 1940, 8: 924.*—(*H. W. H.*)

**Roentgen Therapy for Bronchogenic Carcinoma.**—The results of a study of 250 cases of proved primary bronchogenic carcinoma are prescribed. One hundred and twenty-five

received no roentgen therapy at any time and 125 received such therapy in varying degrees, 102 of these having had less than three courses of treatment with the "massive" technique. Of the nontreated group, not a single case is known to have lived more than a year after proof of his diagnosis (tissue examination following biopsy). Of the 125 cases treated with roentgen therapy, while only 23 were considered to have had complete treatment courses, 25 patients in all lived for from one year to twelve years after the proved diagnosis. The results show not only that roentgen therapy is an excellent method of palliation but also that it has produced so-called cures. The authors believe that any patient who is not in too precarious a physical condition deserves to have at least one course of roentgen therapy; otherwise his life expectancy is, at most, one year. It would seem that, in general, adenocarcinoma is a more favorable type of tumor than epithelioma.—*Roentgen Therapy for Bronchogenic Carcinoma, E. T. Leddy & H. J. Moersch, J. A. M. A., December 28, 1940, 115: 2239.*—(*G. L. L.*)

**Emphysema.**—This study was made in an effort to determine the relationship between pulmonary emphysema and pulmonary vascular changes and their combined effect on the heart. The material for study was selected from cases in which postmortem examination revealed essential emphysema without evidence of hypertension, valvular heart disease or other intrinsic heart disease which could have played a material part in affecting the size and weight of the heart. In the cases studied the heart was examined to determine the weight and the thickness of the ventricles. From these measurements the degree of ventricular hypertrophy and dilatation was determined. The presence and degree of coronary and aortic sclerosis were also recorded. The pulmonary artery and its main branches were studied for gross and microscopical evidence of arteriosclerosis. Only those cases of emphysema which were of considerable magnitude were included in the study. Thirty-two cases of patients from forty to seventy years of age were available. Of this number death from cardiac failure with decompensation occurred in 14, or 44 per cent. In 4 cases death resulted from cardiorespiratory failure during severe attacks of asthma. There was a progressive increase in the occurrence of congestive heart failure as the degree of emphysema increased. In all cases the weight of the heart was significantly more than normal and there was a consistent increase in the degree of right ventricular hypertrophy. In 11 cases there was appreciable hypertrophy of the left ventricle but in none of these was the enlargement of the left ventricle relatively greater than that of the right. In only 6 cases were all the pulmonary arterial divisions normal. In all the others there was some degree of atherosclerosis. The degree of arteriosclerotic involvement of the small muscular arteries increased with the severity of the emphysema. It is felt that in emphysema a demand exists for increased pulmonary circulation to compensate for the defect in ventilation. The heart can be expected to meet this demand only as long as the vascular reserve is not exceeded. Consequently heart failure in such cases results in part from the sclerotic changes in the pulmonary arterial tree. The aetiology of pulmonary arteriolar sclerosis is controversial. In emphysema the arteriolar changes probably are a secondary manifestation of an already present pulmonary hypertension which has been produced by the compression and destruction of the capillary bed.—*Pulmonary Emphysema; A Study of Its Relation to the Heart and Pulmonary Arterial System, R. L. Parker, Ann. Int. Med., November, 1940, 14: 795.*—(*A. A. E.*)

**Obstructive Emphysema.**—Roentgen evidence of obstructive emphysema depends upon whether or not the involved lung or lobe occupies more space than it should at the particular phase of respiration at which the film is taken. At full inspiration there may be no evidence of overdistention, but at the end of expiration the effects of failure of deflation of a lobe or lung may be obvious. In some instances of inferior or middle lobe obstruction the emphysema may be relatively slight; the obstructed lobe may contain approximately the quantity of air it does at the end of a quiet

inspiration. Even on forced expiration, although slightly increased radiability may be present in comparison with the rest of the lung, no limitation or flattening of the diaphragm and no displacement of the mediastinal structures may occur because the relatively increased space occupied by the incompletely deflated lobe may be compensated for by compression of the other normally deflatable lobe or lobes. Two cases, in which the diagnosis of obstructive emphysema was made by the detection of abnormally wide respiratory movements of lower lung structures, are reported by the writer. In Case I (tuberculous ulcer of the right lower lobe bronchus), fluoroscopy disclosed a striking up-and-down movement of calcium shadows in the lower part of the right lung, parallel with and approximately equal to the excursion of the right diaphragm. These shadows did not approach each other as the diaphragm rose on expiration and the right lower lobe did not appear to deflate. A film taken in expiration showed an increased radiability of the right as compared with the left lower lobe. There was no displacement of the heart and no limitation or flattening of the diaphragm. A diagnosis of obstructive emphysema was made. Subsequently, bronchoscopy revealed a mass of tuberculous granulation tissue protruding from the wall of the right lower lobe bronchus. In Case II (foreign body in the right middle lobe bronchus) fluoroscopy showed wide respiratory movement of the lung markings in the region of the right middle lobe. The bronchovascular structures did not approach each other as the diaphragm rose on expiration, and the right middle lobe did not seem to deflate. There was no displacement of the heart or flattening or limitation of the diagphragm. Films taken in expiration and inspiration showed wide displacement of the transverse interlobar fissure and distortion of the right lower lobe bronchial shadows along the heart from pressure of the undeflated middle lobe. A diagnosis was made of obstructive emphysema of the right middle lobe due to foreign body in the middle lobe bronchus. At bronchoscopy a piece of bone was removed from the middle lobe bronchus. Subsequently, fluoroscopy showed the middle lobe to be inflating and deflating normally.—*Abnormally Wide Respiratory Movement of Lower Lung Structures, R. Golden, Am. J. Roentgenol., September, 1940, 44: 325.*—(*W. H. M.*)

**Interstitial and Mediastinal Emphysema.**—A fatal case of pulmonic interstitial emphysema and mediastinal emphysema occurring in a child of twenty-two months and arising from aspirated pieces of peanut is described. Circulatory embarrassment from the pressure of this emphysematous air was the principal cause of death.—*Pulmonic Interstitial and Mediastinal Emphysema, J. H. Fisher & C. C. Macklin, Am. J. Dis. Child., July, 1940, 60: 102.*—(*M. .B*)

**Obstructive Emphysema.**—The clinical and roentgenographic observations on 7 infants and children in whom pulmonary cavities developed during infections of the respiratory tract are reported. The differential diagnosis of pulmonary cavitation is considered, and it is concluded that in these cases the clinical and serial roentgenographic features are consistent with regional obstructive emphysema and are not consistent with pulmonary abscess or congenital cystic disease of the lung. The question is raised whether air-fluid levels in pulmonary cavities are pathognomonic of pulmonary abscess, and it is suggested that in these cases inflammatory exudate in emphysematous cavities caused shifting air-fluid levels similar to those produced by necrotic fluid in necrotic cavities. The pertinent literature is reviewed dealing with the morbid anatomy, the roentgenographic signs and the bronchoscopic observations in regional emphysema and with its causal mechanism, incomplete bronchial obstruction.—*Regional Obstructive Pulmonary Emphysema in Infants and in Children. Emphysematous Cavities and their Similarity to Necrotic Cavities, Congenital Pulmonary Cysts and Loculated Pneumothorax, John Caffey, Am. J. Dis. Child., September, 1940, 60: 586. (Author's Summary)*—(*M. B.*)

**Bronchostenosis.**—Definite localized narrowing of a bronchus is a much more common

complication of either allergic or infectious asthma than is generally believed. The lesion is thought to be primarily inflammatory in nature and not due to allergic oedema or bronchial spasm. As a result of stenosis of the bronchus, movement of air entering or leaving the segment of pulmonary tissue beyond the stenosis is inhibited and bronchial secretions are retained in that part of the lung so that partial or complete atelectasis results. One hundred and forty patients suffering from asthma were examined bronchoscopically and 60 of these were found to have definite stenosis of one or more bronchi. Careful study of these 60 patients revealed the fact that bronchostenosis complicating asthma produces characteristic symptoms and physical and roentgenological signs. A prominent symptom is severe, persistent and sometimes paroxysmal cough which greatly aggravates the asthma. Frequently it is difficult for the patient to raise sputum. When sputum is raised it is usually mucopurulent in character and in 40 per cent of the cases the sputum was blood-streaked. The source of the blood-tinged sputum is the inflammatory tissue in the bronchus and secondary pulmonary congestion. In 68 per cent of the cases febrile episodes with or without chills occurred. This fever was usually sustained for several days and was accompanied by leucocytosis. Uncomplicated asthma is never accompanied by fever, haemoptysis or purulent sputum. Therefore the presence or history of any of these features is strongly suggestive of bronchostenosis. Fifty-three per cent of the patients in this series gave a history of pneumonia and 35 per cent had had pleurisy. Bronchostenosis of itself may produce symptoms and physical signs which are strongly suggestive of bronchopneumonia. The physical signs that accompany bronchostenosis are secondary to the atelectasis which is present. Persistent unilateral physical signs are of most important diagnostic significance because in uncomplicated asthma the physical signs are the same in both lungs. Roentgenological observations in the presence of bronchostenosis are characteristic of those of atelectasis or occasionally of bronchiectasis. However, in 43 per cent of the cases in this series the roentgenograms of the thorax were essentially negative or were not diagnostic of bronchostenosis. When the patient's history or the physical or roentgenological observations suggest bronchostenosis complicating asthma, bronchoscopy should be performed and if a stenotic bronchus can be seen it should be dilated and the retained secretions removed by aspiration. A primary bronchial neoplasm complicating allergic asthma was found in one of the 60 cases. Occasionally more than one stenotic bronchus is found in the same patient. In instances of recurrent bronchostenosis the stenosis may recur in the same bronchus or it may occur in other bronchi of the same or opposite lung. The prevention of recurrences of bronchostenosis depends upon the avoidance of infections of the respiratory tract, postural drainage, and treatment of the underlying causes of the patient's asthma. In all instances of bronchostenosis in this series of cases the stenotic bronchus was in the lower half of the lung, that is, the region to which excessive secretions gravitate. Bronchostenosis was found about equally in males and females; the youngest patient was six years of age, the oldest sixty-four. In half of the patients the asthma had an allergic basis and in the remainder it was infectious. Treatment should be dilatation of the stenosed bronchus through the bronchoscope. Asthmatic patients are quite tolerant of careful and gentle bronchoscopy. In all cases the sputum should be carefully examined for tubercle bacilli. Bronchoscopy rarely should be done during the acute phases of the illness resultant from bronchostenosis. All asthmatic patients with a history or physical signs suggestive of bronchostenosis and those who have had pneumonia or pleurisy in the past should be bronchoscoped in order to verify or rule out the presence of bronchostenosis and to treat any existent bronchial strictures.—*Bronchostenosis Complicating Allergic and Infectious Asthma, L. E. Prickman & H. J. Moersch, Ann. Int. Med., September, 1940, 14: 387.*—(*A. A. E.*)

**Cyst of Lung.**—Recent articles published on congenital cystic disease of the lungs have applied a new name to a well known and long established clinical and pathologic entity. X-rays have permitted the visualization of the cysts in the living subject, but they have added nothing to the excellent clinical descriptions of

the disease made by the older authorities, as far back as the year 1650, under the name of emphysema. Reports of the clinical findings of some authors, notably Laennec in 1846, are so remarkably accurate that they leave no question that cases of congenital malformation of the lungs were observed and well understood long before the advent of the X-ray. Congenital cystic disease of the lungs is more descriptive than pulmonary emphysema, but not as satisfactory as congenital cystic malformation of the lungs. Selecting a new name for an old, well known condition is not only deceiving, but worse, it threatens the loss of the most interesting and valuable literature that has been compiled by our predecessors. We may deceive our successors and limit their perspective as much as we would by failing to recognize that the great advance in our knowledge, that of visualizing the cystic changes in the lungs in life, is due not to the work of Roentgen alone, but to a long line of scientists who devoted their attention especially to electrical research. In a case reported, every detail could be duplicated in the writings of the authors quoted. The only new feature is the visualization of cysts in the living subject, thanks to the development of the X-ray.—*Congenital Cystic Malformation of the Lungs, Formerly Known as "Vesicular or Hypertrophic Pulmonary Emphysema," Recently Described as "Congenital Cystic Disease of the Lungs," With Report of a Case, C. T. Sharpe, Radiology, June, 1940, 34: 692.—(G. F. M.)*

**Cyst of Lung.**—Three forms of pulmonary cysts can be observed: (*1*) cysts without communication with the bronchial tree, (*2*) cysts communicating with the bronchial tree by means of a valvular mechanism, (*3*) cysts with direct communication with the bronchial tree. In a twenty-seven month old boy the diagnosis of a congenital valvular pulmonary cyst of the entire right upper lobe with compression atelectasis of the middle and lower lobes was made. Four years later following a bronchitis a secondary infection of the cyst with formation of pus was observed. After repeated taps and expectoration of some pus, the atelectatic lobes reëxpanded and the cyst disappeared.—*Über die Heilung einer kongenitalen Riesenzyste der Lunge, H. Brügger, Ztschr. f. Tuberk., June, 1940, 85: 14.—(G. C. L.)*

**Pulmonary Embolism.**—Two hundred and forty-seven cases of pulmonary embolism have been reviewed; 166 pulmonary embolisms occurred in medical patients, 80 were postoperative or posttraumatic and one was postpartum. The postoperative incidence in the San Francisco Hospital between 1928 and 1936 was 0.112 per cent. Of 198 consecutive autopsies from July 1939 to February 1940 pulmonary emboli were found in 33 cases (16 per cent). The most mportant factor in thrombosis formation is slowing of the blood flow. Pulmonary embolism was observed between the fourth and the thirtieth day after an operation, the average being the tenth day. Instantaneous deaths were usually due to emboli occluding at least 50 per cent of the pulmonary circulation. Formation of thrombi should be prevented by active and passive motion of the lower extremities, as most postoperative emboli have their origin from thrombi in the iliac and femoral veins. Compared to other forms of treatment of pulmonary embolism, intravenous injections of papaverine give, judging from the literature, the best results; only 12 successfully operated cases have been reported to date.—*Pulmonary Embolism. A Review of the Literature with Additional Statistics Gathered from a Study of 247 Cases, P. R. Westdahl, West. J. Surg., February, 1941, 49: 77.—(G. C. L.)*

**Pulmonary Embolism.**—To eliminate the errors in interpretation of chest roentgenograms resulting from preterminal changes and collapse of lungs after their removal from the thorax, it has become routine at the Massachusetts General Hospital to take erect, posteroanterior and lateral chest teleroentgenograms immediately after death in all necropsied cases. Formalin fixative is injected into the trachea until the lungs are approximately the same size as during life. At the end of one week the specimen is sectioned and an attempt is made to find an anatomical explanation for all shadows on the roentgenogram. Fourteen per cent of 400 cases showed pulmonary embolism or infarction. This represents an increase of over 50 per cent, when compared with 9 per cent for 3,500 autop-

sies at the Hospital before this method of study was started. Forty per cent of the total of 370 cases with embolism and infarction were postoperative patients; 30 per cent were cardiac patients and, interestingly, 30 per cent were noncardiac medical patients, a group in which one does not usually expect pulmonary embolism or infarction. A large percentage of the emboli arise from symptomless thromboses of the deep veins of the legs, usually in the popliteal regions. Roentgenograms of acute fatal pulmonary embolism show no variation from normal, but patients surviving massive embolism may show a pleural effusion. Massive pulmonary embolism with subsequent organization and superimposed thrombotic extension can produce a *cor pulmonale*. Infarcts are always peripheral in location, that is, one or more pleural surfaces are invariably involved. The most common sites are at the costophrenic angles, at the anterior and posterior lung margins near the mediastinum, or at the margins of the middle lobe. There are usually several infarcts in each lung, the average size being 3 x 2 x 1 cm. Their roentgen shadows may simulate localized pleural thickening. The first evidence of an infarct does not usually develop before twelve hours and occasionally not before twenty-four hours after the onset of symptoms. In few instances is the shape of the lesions triangular, and, contrary to textbooks, when a roughly pyramidal or triangular shadow occurs, the apex of the triangle is directed away from the heart instead of toward it. The cardiac margin of the infarct tends to be rounded. When more than one infarct occurs, summation shadows of nearly any shape may be produced. The use of the Potter-Bucky diaphragm and of different projections will "dissect" the shadows into their component parts. In bronchogenic carcinoma, all of the area supplied by the afected bronchus shows the same general density, whereas in infarcts, normally aerated lung can be demonstrated between multiple, sharply defined, peripherally placed areas of consolidation involving one or more lobes. An infarct of the middle lobe may be mistaken for atelectasis or interlobar pleural fluid; however, the sharply outlined shadow of middle lobe infarct, with horizontal superior margin and convex inferior margin, does not extend to the lung root or axillary line. Pleural effusion was present in 40 per cent of the cases, but in only one-third of the group could a causal relation be inferred between infarction and the occurrence of pleural fluid. The writers believe that if there is no evidence of active tuberculosis in the lungs, and excluding apical adhesions, the presence of a "dry" pleuritis (linear shadow on the roentgenogram) or pleuritis with a sterile effusion is more likely to be infarction than tuberculosis. The term "incomplete infarction" is suggested for the syndrome characterized by pleural pain or blood spitting or both, associated with a rapidly appearing and disappearing infarct-like area of consolidation in the lung without alveolar wall destruction. There may be no pleural pain, blood spitting or any obvious source of an embolus, but given any one of these findings, together with roentgenological evidence consistent with an infarct, the diagnosis may be made. In view of the fact that one-third of the cases of pulmonary infarction occur in noncardiac medical patients it is important to consider pulmonary infarction in the differential diagnosis of pulmonary disease in ambulatory patients.—*Correlation of Post-Mortem Chest Teleroentgenograms with Autopsy Findings, A. O. Hampton & B. Castleman, Am. J. Roentgenol., March, 1940, 43: 305.*—(*W. H. M.*)

**Acute Pulmonary Oedema.**—A physiological explanation for the efficacy of positive pressure respiration in acute pulmonary oedema is offered. Meltzer in 1904 produced pulmonary oedema in rabbits by the intravenous injection of adrenalin. In 1909 Emerson showed that pulmonary oedema so produced could be consistently removed by applying artificial respiration through a tracheotomy tube. But since 1878 it has been known that positive pressure could be used as an aid to respiration. Pulmonary congestion and oedema may also be caused by tracheal stenosis. In this latter case stenosis causes an immediate increase in the intrapleural negative pressure. As the intrapleural pressure becomes more negative blood enters the lungs from the right ventricle more readily but the passage of blood into the left auricle is hindered; even more difficult is the passage of blood from the left ventricle into the extrathoracic aorta. A progressive accumula-

tion of blood in the lungs follows and with this congestion there is an increase in the capillary blood pressure causing a greater filtration pressure in the direction of the alveoli. Finally the high intrapleural negative pressure exerts a suction action on the capillaries resulting in exudation of serum into the alveoli. In another group of cases pulmonary oedema may be caused by increased permeability of the capillary walls resultant upon the lack of oxygen that may accompany pneumonia and similar pulmonary afflictions. Congestion and oedema consistently follow inspiratory rather than expiratory obstruction. The experiments upon which the conclusions are based were carried out in rabbits anaesthetized with evipal or ether. Adrenalin was injected into the ear veins in a dosage of 0.5 cc. of a 1:1,000 solution per kilogram of body weight. In almost all instances this produced massive pulmonary oedema which was quite similar to that observed in humans in left ventricular failure. In the animals that were allowed to die as a result of the injection of the large dose of adrenalin, the heart was found to be greatly enlarged, the enlargement being due to dilatation of the right ventricle. The left ventricle was contracted and the lungs contained large amounts of frothy, blood-tinged fluid. When positive pressure was applied by means of intermittent inflation of the lungs, pulmonary oedema could be prevented. It was found that the effect of positive pressure respiration was to raise the intrapleural pressure so that it was atmospheric at one phase of respiration and slightly negative at the other phase. The increased intrapleural pressure diminishes the flow of blood into the right ventricle and hence into the lungs. This causes lower capillary blood pressure and hence lessens the tendency for corpuscles and serum to pass through the capillary wall. In 10 normal humans during positive pressure respiration there were no significant changes in the pulse rate, respiratory rate, or in the systolic and diastolic arterial pressures. There were, however, a consistent elevation of the venous pressure and prolongation of the circulation time. In 14 patients with congestive heart failure, the only significant change caused by positive pressure respiration was an elevation of the venous pressures. The venous pressures were more elevated in the patients with congestive heart failure than in the normal controls. There are presented the histories of 7 patients with acute pulmonary oedema in whom positive pressure respiration caused prompt disappearance of the oedema. Positive pressure is maintained by means of a motor blower unit which can maintain a pressure of 10 cm. of water. In most instances a pressure of 3 to 7 cm. is used. The function of positive pressure in acute pulmonary oedema is to exert a direct opposing physical force on the external capillary wall tending to counteract the tendency to ooze serum. It also decreases the amount of blood which enters the right heart thereby diminishing pulmonary congestion and allowing the heart to function on a smaller volume of blood.—*Positive Pressure Respiration and Its Application to the Treatment of Acute Pulmonary Edema, A. L. Barach, J. Martin & M. Eckman, Ann. Int. Med., December, 1938, 12: 754.*—(*A. A. E.*)

**Oedema of Legs following Strapping of Chest.**—Two cases are presented showing the development of oedema of the feet and legs after fixation of the chest wall for fractured ribs. The first case, a man aged sixty-three, sustained an injury to the anterior chest. An X-ray film revealed fracture of the third rib on both sides, 1½ inches from the anterior end. Adhesive strapping was applied to the whole circumference of the chest from the third rib downward. The patient was observed regularly. Three weeks after strapping he complained of swelling of both ankles. Physical examination revealed marked pitting oedema of both feet and legs. There was no oedema elsewhere. There were no varicose veins of the legs. The heart was completely normal and the blood pressure 120/80. Urine examination was negative. There was no ascites, splenomegaly, hepatomegaly, lymphadenopathy nor dyspnoea. The strapping was removed and the swelling disappeared completely within forty-eight hours. The second case, a man aged fifty, was also strapped for fractured ribs. After wearing the strapping for one month oedema of the legs and feet appeared. The findings on physical examination were entirely negative except for the oedema; there was normal blood pressure, no abnormal cardiac signs, no previous history of

swelling anywhere, no abnormal cardiac or renal history, no varicose veins. The oedema completely cleared within two days after removal of strapping and, as in the previous case, had not recurred two weeks later. No similar cases were found in the literature. The possible mechanism of the oedema is ascribed to inadequate deep inspiration which embarrasses the venous return of the circulating blood.—*Bilateral Oedema of Legs and Ankles following Fixation of Chest for Fractured Ribs, B. Hirsh & R. Ellis, Brit. M. J., November 2, 1940, 2: 593.*—(*D. H. C.*)

**Lipoid Pneumonia.**—That aspiration of fats and oils into the lungs may give rise to a special variety of pneumonitis was determined experimentally as early as 1920, but only in recent years has it been realized that lipoid disease of the lungs occurs sufficiently often to make it a relatively important pathological and clinical entity and the number of cases reported is steadily increasing. Liquid petrolatum is most often responsible because of its wide employment. Masses of phagocytes invade the collections of oil in the alveoli, penetrate the alveolar walls, enter and obstruct the lymphatic vessels and form tumefactions like paraffinomata. Fibrosis with variable degrees of contraction constitutes the final stage. At any stage, bronchopneumonia from infection may be added. Symptoms and physical signs of mild uncomplicated lipoid disease are meager, but the roentgenographic manifestations accurately reflect the morbid anatomical changes. When bronchopneumonia is superimposed, the lipoid factor is likely to be overlooked both clinically and roentgenologically.—*Lipoid Pneumonitis, B. R. Kirklin, Radiology, September, 1940, 35: 261.*—(*G. F. M.*)

**Unexpandable Lung in Empyema.**—The failure of the lung to expand following surgical drainage of postpneumonic empyema may be due to bronchial obstruction caused by retained secretions. The aetiological agent in the genesis of such atelectasis is apparently the presence of thick mucus in the smaller bronchioles and alveoli. In 2 cases reported by the writer and 5 reported by Butler, the patients' ages ranged from three to seventy years. In 4 cases the empyema complicated lobar pneumonia of the left lung and in 3 it followed a similar process on the right. Bacteriological studies of the pleural exudate in 5 cases showed pneumococci in 2, pneumococci and streptococci in 2, and *streptococcus viridans* in one. In all 7 cases the empyema was treated by preliminary aspiration followed by rib resection drainage. The postoperative convalescence in all 7 cases was poor. After the initial drop in temperature which followed surgical intervention for the empyema, there was a secondary rise usually occurring during the latter part of the first postoperative week. In the 2 cases presented in this article, postoperative roentgenographic studies of the chest showed incomplete expansion of the lung, infiltration of the affected lung and a residual collection in the pleural cavity. Serial roentgenograms over a period of weeks failed to show any progress in the degree of pulmonary expansion after the initial immediate postoperative expansion. Because of the altered intrathoracic status (pneumonia, empyema, surgical opening into the pleural cavity and the presence of some degree of pneumothorax) the changes accompanying postoperative atelectasis, such as mediastinal retraction, elevation of the diaphragm and contraction of the intercostal interspaces on the affected side, are not elicited in these cases. In the author's cases there was rapid and progressive improvement after bronchoscopic manipulation or expectoration of the thick blood-streaked mucus. In all of these cases the patients felt considerably better after this had taken place—they were able to breathe more easily, the temperature began to drop and the lung began to expand soon thereafter. The uniform course in all these cases warrants the observation that in any case of postoperative pneumonic empyema which assumes an atypical course and which is associated with incomplete expansion of the lung the presumptive diagnosis should be bronchial obstruction. In the light of bronchoscopic observations it appears that the atelectasis can be attributed to retained secretions in the bronchioles and alveoli.—*Causes of Failure of Lung Expansion following Thoracotomy for Acute Postpneumonic Empyema, L. A. Hochberg, Am. J. Roentgenol., August, 1940, 44: 178.*—(*W. H. M.*)

**Radiation Changes in Lungs.**—A case is reported of bronchogenic carcinoma in a patient living sixteen years after the onset of symptoms. The patient was treated by deep roentgen irradiation over a period of nine years in several institutions, receiving a total of about 18,300 r. Frequent roentgenograms showed slowly progressive fibrosis of the right lung, pleural thickening, marked elevation and fixation of the diaphragm, narrowing of the intercostal spaces and retraction of the mediastinal structures to the affected side. This syndrome following prolonged deep irradiation is almost pathognomonic of roentgen pleuropulmonitis. During the first several years haemoptysis was frequent and often of alarming proportions. Irradiation aided in stopping these haemorrhages which had their origin in intrabronchial granulations. Death occurred from bronchopneumonia in the opposite lung due to *staphylococcus aureus* with renal abscesses and septicaemia. Necropsy revealed the right lung converted into a structure of fibrous consistence, the size of a fist. The pleura was about 4 mm. thick and of almost cartilaginous consistency. Atelectasis was present wherever there was enough lung structure to permit this to occur. Long search disclosed no evidence of carcinoma until study of serial sections revealed an epithelial tumor 2 cm. in diameter with necrotic center and viable periphery lying imbedded in a dense mass of fibrous tissue just outside the hilum. The growth apparently arose from the branch of the right bronchus leading to the upper lobe. The mediastinal lymph nodes contained no tumor. There were marked obliterative changes within the branches of the right pulmonary artery.—*The Deleterious Effects of Deep Roentgen Irradiation on Lung Structure and Function, V. C. Jacobsen, Am. J. Roentgenol., August, 1940, 44: 235.*—(*W. H. M.*)

**Radiation Reaction in Lung.**—Experimental work on rabbits reveals pulmonary changes commencing one to two hours after irradiation and lasting one to seven days. These consist of degeneration of lymph follicles, bronchorrhoea, hyperaemia, transudation and leucocytic infiltration. After a latent period of two to three weeks, there is a marked outpouring of leucocytes, reaching maximum one to two months after the irradiation. As this subsides macrophages and giant cells appear. The end stage consists of proliferation of connective tissue, sclerosis, bone production and slight proliferation of bronchial epithelium. Roentgen evidence of radiation changes in the lungs were found in 36 of the present series of cases. Cough and occasionally fever are expressions of lung involvement. Physical examination does not often indicate infiltration. Roentgenographic examination reveals irregularly spotted or striped shadows; less often there is a more diffuse infiltration. The striped shadows may radiate from the hilum in the shape of a fan. The shadows disappear completely in a few weeks, but in late stages there reappear sharply outlined spots and stripes caused by fibrosis. This may be associated with retraction of the chest on the involved side. Twenty-one of the 36 cases were in patients with carcinomata of the breast, 10 had oesophageal carcinoma, 4 were treated for pulmonary metastasis, and one had a primary carcinoma of the lung. The frequency of radiation reaction in the lung in carcinoma of the breast was 5.4 per cent (21 cases in 386); for carcinoma of the oesophagus it was 20.4 per cent (10 cases in 49). It is concluded that in irradiation of the thorax for various malignancies, lung changes are likely to develop if epidermicidal doses are administered, especially when there are several portals.—*Pulmonary Lesions after Roentgen and Radium Irradiation, R. B. Engelstad, Am. J. Roentgenol., May, 1940, 43: 676.*—(*W. H. M.*)

**Radiation Reaction in Lung.**—Studies were made on 18 cases exhibiting changes in the lungs and thorax following roentgen therapy. The earliest roentgenographic change is a haziness of the lung in the irradiated area. This is soon followed by the appearance of irregular patches of consolidation of uneven density which radiate from the hilum. Such changes may appear after a few massive treatments or after a single course of irradiation of high total dosage over fields of moderate size. When portals are large, concomitant pulmonary and pleural reactions are the rule. Adhesions develop between pleura, pericardium and diaphragm. Pleural, pericardial and less frequently interlobar effusions are occasionally present. If extensive

injury is not produced, the sole residue months later may be slight pulmonary fibrosis and a few adhesions between pleura, pericardium and diaphragm. In instances of extensive injury in which late fibrosis is marked, roentgenograms disclose atelectasis, retraction of heart and trachea, contraction of the thorax, marked pleural thickening and tenting of the diaphragm. Uninvolved portions of the lung undergo compensatory emphysema. With the passage of years, large, sharply outlined, calcific plaques may appear in the irradiated lung or pleura. In patients surviving for long periods after treatment is given, the ribs show narrowing, irregularity in contour and osteoporosis. Sharply circumscribed circular areas of bones of absorption are present. Fractures, in the absence of trauma, are common in the irradiated ribs or clavicle. Where bone displacement is slight, healing may be by bony union, but nonunion is frequent; the ends of the bone become eburnated, resulting in pseudarthroses. In all cases of lung damage, the overlying skin reveals some atrophy and telangiectasis. More extensive radiation changes are indicated by induration of skin and underlying tissues, often stony hard, with resultant contraction of the chest wall. Symptoms such as cough, slight dyspnoea, localized chest pain, and less often fever and sweating are usually transient. In cases of extensive lung damage, cough, dyspnoea and chest pain may become severe and may be accompanied by chills, fever and haemoptysis. As a result of the extensive pulmonary fibrosis, the terminal clinical picture may be that of right heart failure. Postmortem examinations were done on 8 of the 11 patients who died. Hypertrophy or dilatation of the right heart was present in 6. The involved portions of the lungs were atelectatic and fibrotic; adhesions were often present. Microscopy in cases given prolonged irradiation showed severe fibrosis and hyalinization accompanied by bronchitis and bronchiectasis; persisting alveoli were small and frequently filled with inflammatory exudate; acute cases showed areas of oedema, haemorrhage and necrosis.—*Post-irradiation Changes in the Lungs and Thorax, J. R. Fried & H. Goldberg, Am. J. Roentgenol., June, 1940, 43: 877.*—(*W. H. M.*)

**Radiation Reaction in Lung.**—By histological study of the lungs of an unselected series of 398 autopsied cases of malignant disease, primary between diaphragm and neck, it has been possible to collect 29 cases (7 per cent of the entire group, 12 per cent of 234 irradiated cases) which showed changes referable to irradiation of the lung. An erroneous diagnosis of radiation reaction was made in 3 cases which it was found later had not received irradiation to the chest. In another series of 1060 autopsies, there was a 99.3 per cent accuracy in diagnosis of radiation pneumonitis based solely on histological changes. The salient points used in differentiating radiation reaction from other lung inflammatory changes, such as chronic nonspecific interstitial pneumonitis, influenzal pneumonitis, chemical pneumonitis are: (*1*) a hyaline membrane closely adherent to alveolar walls and never present alone but always associated with one or all of the following changes; (*2*) swollen alveolar lining cells with or without desquamation and fibrosis, oedema in the supporting tissue; (*3*) oedema; (*4*) diffuse alveolar fibrosis without evidence of organizing pneumonia, tuberculosis, or silicosis; (*5*) an unusual fibrillar hyalinization of alveolar walls; (*6*) hyalinization of arterial walls; (*7*) interalveolar capillary changes such as swelling of endothelium and thrombosis. It is probable that certain stages of radiation response have not been recognized histologically and may be of such a nature that differentiation from other disease processes is impossible. Depending on the time of appearance and nature of the above pathologic changes, three stages are present: acute, late, and late with superimposed acute reaction. In only 62 cases were chest roentgenograms suitable for study. In only 5 (8 per cent) were there roentgenographic changes definitely due to irradiation. It seems fair to assume that this is not a true index of the frequency of pulmonic reaction. Were radiation changes watched for from the time of beginning therapy, study, both antemortem and at autopsy, would give a higher percentage of cases showing reaction. The earliest roentgenographic change seen was an increase in radioluscency of the chest. This may be followed after an interval of a few weeks by an increased density, part of

which is apparent as discrete bands, with displacement of the trachea, heart and mediastinal shadow toward the affected side. This early reaction can be expected in some cases to show complete regression and in others to show a residual fibrosis of the lung with or without pleural fibrous adhesions, depending upon the degree of injury and subsequent treatment. Late contraction of the thoracic cage is due in part to decrease in size of the lung and, in part, to the pleural adhesions. By means of serial films it is usually possible to differentiate the development and passing of a radiation reaction from the presence of metastatic disease. Variables such as age, the thickness of the chest wall, amputation of the breast, the existence of pulmonary metastases, the existence of pneumonia, were considered and found to have no significant relation to the development of radiation changes. The amount of radiation alone does not determine the development of a radiation reaction, although the proportion of lungs showing changes increases with the amount of irradiation. The majority of cases showing radiation reaction present symptoms of an unproductive hacking cough during the height of the local skin reactions. Dyspnoea and chest pain are present less frequently.—*Radiation Reaction in the Lung, S. Warren & J. Spencer, Am. J. Roentgenol., May, 1940, 43: 682.*—(*W. H. M.*)

**Status Asthmaticus.**—Death is rare in *status asthmaticus*. Seven patients who died in *status asthmaticus* and came to autopsy are reported. In all, the autopsy findings were consistent with bronchial asthma and no extrapulmonary cause of death was present. The patients were all women with a mean age of forty-five. The duration of the fatal attack was variable, ranging from two days to several weeks. In every case there was great respiratory distress with difficulty in expiration. In 5 cases the heart was normal on physical examination; in one there was cardiac enlargement and in the seventh, there was a shifting rate. A few of the cases manifested signs of right heart failure. Blood pressure was normal in all cases, and fever was observed in 4 cases. The eosinophiles were increased in only 2 patients. In the terminal stages, the patients failed to get adequate relief from epinephrine. At autopsy, the lungs were feathery and voluminous filling the pleural cavity. Cut surfaces showed a large amount of thick, tenacious, mucoid material in the middle-sized bronchi. Microscopical sections showed emphysema in all cases. The severest lesions were in the middle-sized bronchi which, for the most part, contained thick plugs showing eosinophiles, Curschmann's spirals and Charcot-Leyden crystals. The bronchial mucosa was thrown into folds and frequently small sacculations of the mucosa could be seen. The mucosa and submucosa were infiltrated with lymphocytes and varying numbers of eosinophiles. In all cases, the submucosal layer was widened and the basement membrane was thick and hyaline. Hypertrophy of the bronchial musculature was present in every case but varied greatly in extent and amount. Inspissated mucus distending the bronchial glands, with pressure atrophy of the acinar cells, was frequently seen. Two patients had bronchopneumonia and in one case there were areas of atelectasis. There was no important cardiac lesion in any of the cases at autopsy. In no case was a relevant, significant pathological condition found in other parts of the body. There is no single criterion for making a pathological diagnosis of bronchial asthma. However, asthma does produce a characteristic picture in the bronchial wall, including a widened basement membrane, infiltration of eosinophiles, a widened submucosal layer, hypertrophy of muscle, and "degenerative" mucous glands. The value of therapeutic agents in *status asthmaticus* is uncertain. A wide variety of drugs was used in this series. The decreasing efficacy of epinephrine is an important observation in these cases. It has been stated that morphine and digitalis are dangerous drugs in the treatment of asthma. Most patients in this series received both drugs; no conclusions could be drawn with regard to the possible harmful effect of these drugs. The voluminous bulging lungs suggest a decreased negative pressure in the thorax. This acts to impede the return of venous blood to the right heart and may partially explain the signs of cardiac failure occasionally seen in *status asthmaticus*. This

suggests that these signs of failure are due to extracardiac, rather than cardiac factors.—*Fatal Bronchial Asthma, B. Craige, Jr., Arch. Int. Med., February, 1941, 67: 399.*—(*H. R. N.*)

**Respiratory Allergy.**—A certain number of patients with severe asthma that occurs during July and early August have been found to be allergic to the spores of rust and smut. Smut fungi occur most commonly on cereals and grasses. The rust spores infest the stems of various grains, grasses and fruit trees. The spores of both these fungi are disseminated by the wind and are particularly numerous throughout July and August. The rust spores reached a high peak about the middle of July, as did those of the smuts. Both persisted through the early part of August. The concentration is higher than that of timothy but not as high as that of ragweed. One hundred and six consecutive patients with asthma and nasal allergy were examined. Seven of these had symptoms at no other time than late July and early August. Ten others suffered severe exacerbations of perennial asthma during that time and 2 had exacerbations of nasal catarrh in late July and early August. The other 87 patients had no symptoms at the time in question. There was thus a definite parallelism between the severity of symptoms and the concentrations of rust and smut spores in the air. Seven patients whose symptoms were confined to July and August gave strong skin reactions to solutions of the spores, but their reactions to pollens and the other fungi were less pronounced. Twelve patients who had periodic exacerbations at the rust and smut season reacted positively to skin tests for these allergens. In the control group of 87 patients, the reactions to rust and smut were negligible. In 2 cases asthmatic attacks were rapidly induced by inhalation of rust. The architecture of the spores, their absence in the air during a large part of the year, the suddenness of the spore concentration in the air, and the high peak which is reached are all factors which support the theory that these spores cause definite respiratory allergy.—*Rust and Smut, Major Causes of Respiratory Allergy, G. L. Waldbott & M. S. Ascher, Ann. Int. Med., August, 1940, 14: 215.*—(*A. A. E.*)

**Oral Pollen Therapy.**—Because of previous conflicting results of the published reports and importance of oral pollen therapy in the treatment of ragweed pollinosis, a coöperative investigation was begun in 1939 by the allergy clinics of 4 medical schools in the midwest. The patients selected were those who had ragweed hay fever or hay fever and asthma and either had never been treated hypodermically or had had no treatment later than 1937. Those treated preseasonally took single doses twice weekly, beginning with 500 pollen units and doubling each dose until 180,000 units was reached. During the season capsules were taken once daily. Those who began treatment during the season took the pollen three times daily. Identically colored capsules containing starch only were given for placebo purposes. In the University of Illinois Clinic 103 patients were studied. Of 39 patients with fall pollen asthma, who were treated orally, 82 per cent were unimproved, and 18 per cent had moderate improvement, but none were completely relieved. Of 32 patients with uncomplicated hay fever, who received oral treatment, 62 per cent were unimproved, and the remaining 38 per cent showed slight to good improvement. Of 32 patients who received placebo capsules only, 78 per cent were unimproved, while 22 per cent reported slight to excellent results. Therefore the results for subjects with uncomplicated hay fever treated with oral pollen show no substantial difference from results for those receiving placebo capsules. In the Rush Medical College Clinic, treatment results were compared of a group of 32 patients given oral pollen capsules with a group of 25 patients treated parenterally. The average daily dose of oral pollen given was fifty times that of the parenteral pollen extract dosage. Of the oral group 25 per cent had moderate to severe gastrointestinal reactions. No systemic reactions occurred in the parenterally treated group. Of the oral group 25 per cent reported improvement as compared with 56 per cent of those treated parenterally. In the University of Michigan—Northwestern University series, 27 patients received oral pollen therapy and all but one also received placebo injections. None received oral placebos. Twelve patients had asthma with their hay

fever. In 12 cases there were gastrointestinal reactions. In 23 of the series there was no indication of benefit. In the other 4 or 14.8 per cent, nasal symptoms were improved, but asthmatic symptoms remained unimproved. The pooled results of all the clinics showed that in 29.8 per cent of 141 cases there was some improvement under oral pollen therapy, but of these cases, only 1.4 per cent had excellent results, 8.5 per cent good results, and 19.9 per cent slight to moderate improvement. Moreover, the oral treatment showed to least advantage in the relief of asthma. The results in the placebo group were surprising and detract substantially from the significance of the percentage results with oral pollen therapy, if not wholly nullifying these results. The appraisal of oral therapy by the authors varies from complete rejection to limited acceptance. Gastrointestinal reactions are to be expected frequently, but are not dangerous. In the control of asthma, the effectiveness of oral therapy is definitely inferior to that of parenteral injection.—*Oral Pollen Therapy in Ragweed Pollinosis: A Cooperative Study, S. M. Fineberg, F. L. Foran, M. R. Lichtenstein, E. Padnos, B. Z. Rappaport, J. Sheldon & M. Zeller, J. A. M. A., July 6, 1940, 115: 23.*—(*G. L. L.*)

**Chest Injuries.**—Patients requiring immediate surgery for war wounds of the chest suffer from a lack of oxygen in the tissues, the anoxia of shock. If, in addition, the pleural cavity is open a further degree of anoxia ensues due to the embarrassment of respiratory exchange. Experiments have shown that wound shock, through the direct action of nervous impulses and tissue metabolites on the respiratory enzyme, reduced cytochrome, interferes with cellular respiration and oxidation. It has also been shown that morphine and anaesthetics act on another respiratory enzyme, dehydrogenase, and produce oxygen starvation of the tissues by interfering with cellular respiration. For this reason the use of morphine and its derivatives is contraindicated in chest wounds requiring immediate surgery. The form of anaesthetic used must combat the existing anoxia and in the case of the open chest relieve the patient of the necessity of increased respiratory effort until the pleural cavity can be closed and the mediastinum and lung stabilized. The closed circuit method anaesthesia is unsuitable because it produces further anoxia. Spinal anaesthesia is not advised because of the fall in blood pressure. Intratracheal insufflation anaesthesia is the method of choice. Because there is a real risk of damaging alveolar tissue at high pulmonary pressure, it is advised that such pressure not exceed 12 mm. of mercury. Nitrous oxide and cyclopropane are theoretically ideal anaesthetics but too expensive. Ether and chloroform are good anaesthetics. A postoperative course of oxygen is advised.—*Anaesthesia in Chest Injuries, J. Halton, Lancet, November 30, 1940, 2: 675.*—(*J. B.*)

**Chylothorax following Trauma.**—A thirty-two year old collier ate a large breakfast of bacon and eggs at six a.m. and while working underground at nine-thirty sustained a fall on his dorsal spine. He experienced an immediate feeling of constriction in the chest. Examination two hours later revealed slight shock but no dyspnoea. X-ray the following day revealed possible damage of the ninth thoracic vertebra, chest X-ray film was normal. Two days following the injury patient had a fever of 100°F. and there was some dyspnoea. The next day the patient collapsed, the pulse rate was 130, temperature subnormal and respirations were rapid and distressed. A moderate amount of pleural effusion on the right was aspirated with removal of a large quantity of fluid resembling pale tomato soup. The fluid proved to be chyle on examination. There was slight temporary improvement in symptomatology but repeated taps were necessary. In all, 27 pints had been removed in the nine days following the discovery of the effusion. Despite extensive supportive therapy, including intravenous glucose infusions and rectal feedings, patient died in a state of exhaustion eleven days after his injury. Postmortem examination revealed a completely collapsed right lung with some chylous effusion around it. The left lung was normal. The parietal pleura along the right side of the ninth and tenth thoracic vertebra (which were fractured) was torn to form a diamond-shaped gap 3 x 3/4 inches.

The anterior spinal ligaments were completely torn across and the thoracic duct was severed as it passed upwards along the spine in close proximity to the pleural tear.—*Traumatic Chylothorax, C. J. Cellan-Jones & W. Murphy, Brit. M. J., November 2, 1940, 2: 590.—(D. H. C.)*

**Industrial Lung Disease Disability.**—In the evaluation of claims for compensation due to pulmonary disability acquired in industry, the final analysis must be made by considering all clinical data in conjunction with tests for functional capacity. Fundamentally, the production of pulmonary fibrosis and emphysema in varying degrees determines the degree of disability. In all clinical considerations individual susceptibility is an important factor, involving constitutional variants in the self-cleansing function of the lung, and other factors such as age, previous infections and nonindustrial dust exposure. Failure in varying degrees of the self-cleansing function of the lung provokes a chronic productive inflammation resulting in pulmonary fibrosis, and accompanied by progressive pulmonary emphysema, at first of compensatory character, but eventually a source of decompensation. From the clinical and roentgenological points of view, pulmonary fibrosis may be classified in the following forms: (*1*) massive (pneumonic subacute; this is seen in acute silicosis and after radium and roentgen ray exposures), (*2*) diffuse interstitial, (*3*) whorled nodular and (*4*) conglomerate (silicosis with infection and anthracosilicosis). At times fibrosis predominates and at others emphysema is the conspicuous factor. Conglomerate fibrosis is more likely to give extensive emphysema. Pulmonary function may be disturbed in 3 ways: (*a*) in ventilation, (*b*) in gas exchange and (*c*) in pulmonary circulation. Functional tests for disability are based upon these 3 pulmonary functions. Maximal breathing capacity determinations measure the degree of ventilation. Impairment of gas exchange is determined by spirometric measurements of oxygen intake and carbon output, and is expressed in 3 ways, by measurement of (*a*) the oxygen debt, (*b*) the oxygen deficit and (*c*) the degree of unsaturation of the haemoglobin of the arterial blood. The efficiency of the pulmonary circulation is determined by measurements of the delay in circulation through the lungs. These 3 functions of ventilation, gas exchange and circulation may be tested best for practical purposes by the so-called exercise tolerance test, the standard employed being 30 steps per minute on a 20 cm. high pedestal, with the patient connected with a spirometer. A patient dyspnoeic on the aforementioned standard exercise, for which he needs 60 per cent or more of his maximal breathing capacity, is recognized as functionally handicapped. The exercise tolerance test is, then, essentially a measure of dyspnoea. The author offers a proposed disability grading classification as follows: (*a*) *Phase of Compensation*, in which fibrosis and emphysema are present to a considerable degree, and apparent by roentgen examination, physical signs and spirometric tests, but which do not as yet restrict the capacity for moderate exercise; (*b*) *Latent decompensation*, fibrosis and emphysema of an extensive degree, tolerated during rest, but readily producing dyspnea on moderate exercise; (*c*) *Manifest decompensation*, with symptoms of functional insufficiency while the patient is at rest.—*Clinical Evaluation of Disability in Pulmonary Diseases of Industry, E. Mayer, J. A. M. A., January 11, 1941, 116: 97.—(G. L. L.)*

**Acute Silicosis.**—A twenty-six year old man was employed in 1936 as a sand-blaster for three months, four hours a day. Beginning January, 1937, he worked at sandblasting all day in a small room heavily laden with silicon dioxide dust and with inadequate protection. Symptoms began during the summer of 1937. Roentgenograms for three consecutive years demonstrate silicosis developing in eleven months and becoming an advanced case in thirty-nine months.—*Acute Silicosis, W. D. McNally, J. Indust. Hyg. & Toxicol., January, 1941, 23: 45.—(T. R. J.)*

**Pneumonoconiosis from Fuller's Earth.**—Roentgenological examination of 49 men who had been working with Fuller's earth from one and three-quarters to sixteen years revealed only 3 normal chests. The remainder all demonstrated some abnormality, the most noticeable finding in the films being a slight increase in bronchial markings. The degree of

physical disability was greater as compared to the same degree of roentgenological changes found in soft coal miners.—*Severe Pneumoconiosis Caused by Inhalation of Fuller's Earth, W. D. McNally & I. S. Trostler, J. Indust. Hyg. & Toxicol., March, 1941, 23: 118.*—(*T. R. J.*)

**Settling Characteristics of Dusts.**—The Leitz tyndallometer was used in an investigation of the rate of settling of different kinds of dust. The relative rates were established in the following order starting with the slowest: two varieties of impure quartz, sericite, pure quartz, crude sulphide ore, kaolin, coarse calcined gypsum ground in buhrstone mills, shale, limestone montmorillonite, "low limestone", magnetite, fine calcined gypsum ground in ball mills. The addition of calcined gypsum to pure quartz produced a dust whose rate of settling was very much faster than that of pure quartz alone. All mixtures of calcined gypsum with quartz settled much more quickly than mixtures with any other materials that were investigated. The existing theoretical laws of settling did not explain the different rates. Although particles suspended in air acquired specific and measurable electrostatic charges, the flocculation and rapid settling was not consistently associated with the attraction of opposite charges. The particle size and the specific gravity appeared to be unimportant factors. The shape and the capacity to absorb moisture have not been fully evaluated as possible influences.—*Settling Characteristics of Various Inorganic Dusts, L. H. Berkelhamer, J. Indust. Hyg. & Toxicol., September, 1940, 22: 276.*—(*T. R. J.*)

**Fibrosis of Lung in Scleroderma.**—The roentgenographic appearance of pulmonary fibrosis occurring in generalized scleroderma has never before been reported. A survey of the literature reveals only a total of 6 autopsy protocols in which a characteristic diffuse pulmonary fibrosis was described. The pathological changes in diffuse scleroderma are not confined to skin and subcutaneous tissue alone but involve the connective tissue system throughout the entire body. Everywhere the same changes occur, namely oedema followed by sclerosis of the collagenous bundles. This may occur in the connective tissue between the alveoli of the lung. The authors report the hospital admission and case summary of a negress, aged thirty, who had the typical clinical and pathological evidence of generalized scleroderma, and in whom it was possible to demonstrate definite roentgenographic changes due to pulmonary fibrosis. Within both pulmonary fields, exclusive of the apices and the lateral aspect of the bases, there was a diffuse reticular shadow extending from the cardiac border to the periphery. This shadow was dense in the lower halves of the lung fields but thinned out and became strandlike in the subclavicular regions. Bronchography revealed a normal bronchial tree. There was a negative tuberculin reaction and no history of respiratory infections or exposure to dusts, and since a diagnostic pneumothorax and X-ray examination proved that the pulmonary shadows were not due to sclerosis of skin and muscle over the chest, it is assumed that they represented extensive pulmonary connective tissue proliferation as a phase of the generalized sclerodermatous process.—*Scleroderma with Pulmonary Fibrosis, J. R. Murphy, P. Krainin & M. J. Gerson, J. A. M. A., February 8, 1941, 116: 499.*—(*G. L. L.*)

**Thorax in Track Stars.**—The ratio of the depth of the chest to the width at the nipple line was determined in over 22,000 children and adults. This, the so-called thoracic index, was .670 in the normal subject. The average in tuberculous patients was .770. The chest of the new born baby is almost round; by the end of the first year the thoracic index is .780, by the end of the fifth year it is .720. The mean thoracic index in 98 track and field stars was .677, the negro boys having deeper chests than the white boys as in the general population. The mean thoracic index was lower in the track stars than in those who participated in discus-throwing, javelin-throwing or shot-put. Of 24 athletes who placed first, second or third in track or field events, 18 had flat chests with a thoracic index below .700.—*Are Track Stars Barrel-Chested?, S. A. Weisman, Journal-Lancet, December, 1940, 60: 539.*—(*G. C. L.*)

**Hodgkin's Disease and Brucella Infection.**—The authors present a study of the clinical, bacteriological and immunological aspects of 14 cases of Hodgkin's disease. They have been able to isolate *Brucella melitensis* from either the blood or from lymph node cultures in all 14 of these histologically confirmed cases. Although these 14 patients were natives of an area in which Brucella infection is widespread, control studies of a larger group of 67 patients suffering from diseases other than Hodgkin's, involving the lymph nodes, resulted in the isolation of Brucella in only one instance. Furthermore, cultures yielding Brucella have been obtained with the greatest frequency when Hodgkin's disease was actively progressing, rather than when it was subacute or temporarily arrested. A sufficient correlation has been found to exist between the cultural and immunological manifestations and the clinical course of these patients to justify the belief that the Brucella infection directly influences the syndrome of Hodgkin's disease. Maximum clinical improvement has coincided with the presence of serum agglutinins and significant degrees of phagocytosis for Brucella. A definite and often striking decrease in the size of enlarged lymph nodes has been observed after administration of sulfanilamide or sulfanilamide or sulfapyridine. Immune serum therapy has been combined with these drugs, but its effectiveness has not been demonstrated. Recent investigations have shown that Brucella organisms isolated from many of the patients suffering from Hodgkin's disease possess capsules and show significant antigenic differences from the laboratory strains. So far, although Brucella has been recovered from the blood of guinea pigs and mice dead following an injection of a suspension of lymph nodes from patients with Hodgkin's disease, lesions identical with those of Hodgkin's have not been observed, and this phase of the problem is still being investigated.—*The Coexistence of Brucella Infection and Hodgkin's Disease, N. B. Wise & M. A. Poston, J. A. M. A., December 7, 1940, 115: 1976.*—(*G. L. L.*)

**Aneurysm of Pulmonary Artery.**—Two cases of aneurysm of the pulmonary artery are reported. The first is that of a woman, aged fifty-four, who gave a history of progressive dyspnoea for six months. The clinical picture was that of myocardial insufficiency. There was a systolic pulsation and thrill in the second interspace to the left of the sternum; roentgenographic examination showed a prominent pulmonary conus which did not pulsate. The blood Wassermann was strongly positive. Death occurred two months after admission. Autopsy showed a fusiform dilatation of the pulmonary artery throughout its intrapericardial extent. Gross and microscopical examination of both the aorta and pulmonary artery revealed typical syphilitic changes. The second case is that of a male, aged fifty-one, who is still alive and in whom the diagnosis is only suspected. There is a history of an untreated penile sore and the blood Wassermann is strongly positive. There has been evidence of progressive myocardial insufficiency for two years. Roentgenographic examination shows a mass in the region between the left border of the left ventricle and the left border of the aorta; this mass pulsates synchronously with, but less vigorously than, the aorta. The clinical impression is that this mass represents a saccular dilatation of the pulmonary artery at its origin. A review of the literature shows at most 26 cases of syphilis of the pulmonary artery. No symptoms or signs are pathognomonic. In most, the onset is insidious, and is characterized by cough and dyspnoea. Later, evidence of myocardial insufficiency appears. Examination often reveals fullness in the region of the third rib and interspace to the left of the sternum. Pulsations, thrills, and systolic and diastolic murmurs are noted in this area. The blood Wassermann is positive in only 50 per cent of cases. Some develop severe pulmonary fibrosis. The X-ray film may show a globular shadow in the region of the pulmonary conus which pulsates feebly and points anteriorly. The course is usually that of progressive myocardial failure; occasionally there is sudden death.—*Aneurysm of the Pulonary Arteries, W. B. Allan & J. P. McCracken, Am. J. Syph., Gonor. & Ven. Dis., September, 1940, 24: 563.*—(*A. G. C.*)

**Aortic Aneurysm.**—Many pulmonary and pleural complications of an aneurysm of the descending arch of the aorta have been de-

scribed. Symptoms may be caused by compression of the trachea, bronchi or lung, or by direct invasion and rupture of the aneurysm into the lung or pleura. The case reported is that of a fifty-three year old colored male in whom an aneurysm of the descending thoracic aorta invaded the lower lobe of the left lung and produced a large pulmonary cavity. The clinical signs and symptoms of this patient and the roentgenographic appearance of his chest were quite similar to those of pulmonary tuberculosis with excavation of the lower lobe of the left lung. It was not until his death and postmortem examination that the true nature of the disease was recognized. At that time there was found in the lower lobe of the left lung a large cavity which communicated with an aneurysm of the descending arch of the aorta. The cavity was filled with lamellated blood clot and lined by a grayish membrane in which coal pigment could be seen. The aneurysm was of syphilitic origin and no evidence of pulmonary tuberculosis was found.—*Saccular Aneurysm of the Descending Thoracic Aorta with Invasion and Cavitation of the Lung Simulating Pulmonary Tuberculosis, H. L. Katz, Quart. Bull. Sea View Hosp., October, 1940, 6: 79.*—(*A. A. E.*)

**Amyloidosis.**—Amyloidosis is a frequent complication of pulmonary tuberculosis and occurs in approximately 21 per cent of all cases in which autopsy is done. Regression of amyloidosis occurs very infrequently. However, 4 patients are reported in whom amyloidosis was diagnosed by urinalysis and by almost complete absorption of congo-red injected intravenously. In these 4 patients the amyloidosis apparently disappeared as their tuberculosis became cured. Subsequent injections of congo-red and urinalyses showed that the amyloid disease had regressed to the extent that it was no longer clinically diagnosable.—*Regression of Amyloidosis, A. W. Pearlman, Quart. Bull. Sea View Hosp., 1940, October, 6: 92.*—(*A. A. E.*)

**Skin Tests with Extracts from Leprous Spleens.**—Protein extracts of human leprous spleen were made. Skin tests with this substance were performed following the technique of the Mantoux test in normal and in leprous patients. Patients suffering from active leprosy did not react specifically, however. There is no evidence from this study that there is a skin test for leprosy comparable to the tuberculin test in tuberculosis.—*Skin Reaction Tests with Tuberculin-Type Extracts of Leprous Spleens, The Joint Committee on Leprosy Skin Tests, Philippine Bureau of Health and the Leonard Wood Memorial, Internat. J. Leprosy, July-September, 1940, 8: 263.*—(*G. C. L.*)

**Leprolin.**—The test material used in these studies was a boiled purée of Hansen bacilli and human tissue from a leprosy nodule. The authors studied particularly the reaction to intracutaneous inoculation with this material in the two forms of leprosy, *Lepra nervosa* and *Lepra tuberosa*. There occurs first a twenty-four to forty-eight hour tuberculin-like reaction, which is probably not very specific. Similar reactions can be produced with leprolin in tuberculin-sensitive individuals, and filtrates from leprosy nodules will also give such a reaction in cases of leprosy. A suspension of boiled uninvolved tissue from lepers produces no reaction. The significant reaction, which appears almost exclusively in *Lepra nervosa*, is first detected four or five days after the inoculation and when all of the erythema of the early reaction has subsided. The skin is observed to remain brownish and slightly sunken at the test site. By the end of the first week a tiny violet papule appears and gradually increases in size. This reaches its peak and may even ulcerate during the third and fourth week after inoculation. Histological studies at the end of twenty-four hours show the nonspecific reaction predominant and manifested by a marked lymphocytic, leucocytic and eosinophilic infiltration with oedema of the periglandular connective tissue. There is also a peculiar fibrinoid swelling of the connective tissue about the small veins of the corium. Twenty-four hours later there is further infiltration and formation of tiny abscesses with necrosis of the collagen fibres of the corium. Even at the end of the first twenty-four hours there are signs of the more specific reaction. There are well defined foci in which there is marked swelling and loss of staining power of the fibroblast nuclei within a slightly baso-

philic protoplasmic mass. At forty-eight hours these foci have become infiltrated with lymphocytes, the centre of the reaction has become oedematous and small foci are present in which the connective tissue fibres are swollen and beginning fibrinoid necrosis and nuclear degeneration have taken place. After four days there is advanced fibrinoid necrosis with vacuolization of the connective tissue cells. At eight days there are multiple foci or nodules of fibrinoid necrosis and beginning organization by spindle shaped and vacuolated epithelioid cells. There may even be Langhans cells at this time and an accumulation of lymphocytes about the periphery of the foci. By the end of twelve days the epithelioid cells resemble the "Virchow cells" seen in *Lepra tuberosa*. There may also be observed large vacuoles containing doubly refractile lipoid material. At thirty days there is a slight vascularization of the border of the nodules and the tissue is almost indistinguishable from that of the skin lesions of *Lepra nervosa*. The authors made over 200 biopsy studies of the spreading borders and new foci about old healed lesions in the latter condition. The evolution of these lesions is similar to the reactions detailed above with the exception that in the early stage bacilli are frequently observed. This form of leprosy which is very chronic and without visceral involvement seems to depend on the presence in the host of a high degree of immunity. In *Lepra tuberosa* the disease is characterized clinically by a more extensive involvement of the host and by a lack of resistance. This observation correlates well with histological studies. The lesions are diffuse or nodular infiltrations consisting chiefly of large vacuolated, lipoid containing histiocytic cells (Virchow cells) which regularly contain an abundance of bacilli. The authors explain the predominance of *Lepra nervosa*, the more benign form, among the dark races with a predominance of the tuberosa form among the whites on the basis of the fact that the latter are rarely exposed before adult life. They have observed that among whites exposed from early life the nervosa form predominates just as among the blacks. Leprolin tests were made on contacts and a high percentage of positive reactors were found, 70 per cent among the personnel in the institution where these studies were made. All children of lepers were negative at birth, 22 per cent were positive at three years, 53 per cent between three and thirteen years, and 86 per cent of children over thirteen years of age were positive.—*Untersuchungen über den klinischen Verlauf und die histologischen Veränderungen allergischer Reaktionen bei der Lepra, W. Büngeler & J. M. Fernández, Virchows Arch. f. path. Anat., 1939 & 1940, 305: 236, 473 & 593.*—(*C. L. D.*)

**Key to Abstractors**

*A. A. E.:* Adrian A. Ehler, Albany, New York.
*A. G. C.:* A. G. Cohen, New York City.
*C. L. D.:* Charles L. Dunham, Chicago, Illinois.
*D. H. C.:* David H. Cohen, New York City.
*E. R. L.:* Esmond R. Long, Philadelphia, Pennsylvania.
*G. C. L.:* G. C. Leiner, New York City.
*G. F. M.:* Gertrude F. Mitchell, Detroit, Michigan.
*G. L. L.:* George L. Leslie, Howell, Michigan.
*H. M.:* Henriette Marcus, New York City.
*H. R. N.:* H. R. Nayer, New York City.
*H. W. H.:* Harlon W. Harrison, Shreveport, Louisiana.
*J. B.:* Joseph Ballinger, New York City.
*J. S. W.:* J. Stanley Woolley, Liberty, New York.
*M. B.:* Miriam Brailey, Baltimore, Maryland.
*T. R. J.:* Teckla R. Jocz, Grosse Pointe Park, Michigan.
*W. G. C. M.:* W. G. C. Munroe, Saranac Lake, New York.
*W. H. M.:* W. H. Merrit, New York City.

# INDEX OF ABSTRACTS OF TUBERCULOSIS


Aaronson, A. L. See Rubin, S. S., *et al.*, 38
Abelló, J. Cavernoscopy, 124
Abscess, Lung, 11, 12, 125
Absence, Congenital, of hemidiaphragm, 38
Acid-fast bacteria, Receptor analysis of, 105
—— bacterium in swine, 10
Ackermann, A. J. Tuberculosis of stomach, 80
Actinomycosis, 128
——, Pulmonary, 16
Activity, Index of, 111
Acute pulmonary oedema, 152
—— respiratory infection, 138
—— silicosis, 160
Adams, Z. B. Tuberculosis of spine, 75
Adenopathy, Tuberculous, 68
Adolescent females, Tuberculosis in, 48
Adolescents, Tuberculosis in, 48
Adults, Primary tuberculosis in, 54, 55
Agnello, V. Pulmonary lues, 15
Air, Residual, 87, 88
Alexander, J., and Haight, C. Hydatid cyst, 28
Allan, W. B., and McCracken, J. P. Aneurysm of pulmonary artery, 162
Allergy, Respiratory, 158
Allergy-producing properties of paratubercle bacilli, 101
Amberson, J. B., Jr., and Muschenheim, C. Interposition of colon, 1
Amyloidosis, 163
Analysis, Receptor, of acid-fast bacteria, 105
Aneurysm, Aortic, 162
—— of pulmonary artery, 162
Annual surveys, 46
Anthracite and tubercle bacilli, 96
Antigenic depot excised, 105
Antiseptics and detergents, 97
Aortic aneurysm, 162
Apical tuberculosis, X-ray study of, 108
Argemi, J., and Müller, R. W. Pericavitary atelectasis, 4
Army, Tuberculosis in, 49
Artery, pulmonary, Aneurysm of, 162
——, ——, Atherosclerosis of, and pulmonary emphysema, 29
Arthritis, Tuberculous, 73
Asbestosis, Silicosis and, 25
Ascher, M. S., and Waldbott, G. L. Respiratory allergy, 158
Aspergillosis in a skunk, 17
Asthma and tuberculosis, 61
——, Tuberculosis and, 60
Asthmatic bronchitis in children, 141
Asthmaticus, Status, 157
Atelectasis, 32
——, Pericavitary, 4
Atherosclerosis of pulmonary artery and pulmonary emphysema, 29
Atypical bronchopneumonia, 135, 137
Auerbach, O. Tuberculosis of bones, 71
Avian tubercle bacillus, Sensitization of cattle by, 9
—— tuberculin, 9

Babolini, G. Suction drainage of cavities, 7
——, ——., and Bottari, G. Intracavitary drainage, 118
——, ——. See Monaldi, V., *et al.*, 120
Bachmann, E. Follow-up in tuberculosis, 112
Bacilli, paratubercle, Allergy-producing properties of, 101
——, tubercle, Anthracite and, 96
——, ——, bovine, Isolation of, 100
——, ——, Culturing, from sputum, 98
——, ——, from spinal fluids, 100
——, ——, growth of, Inhibition of, 97
——, ——, in laryngeal content, 98
——, ——, —— rats, 84
——, ——, —— spinal fluid, Meningococci and, 81
——, ——, —— swine tonsils, 10
——, ——, —— urine, 99
——, ——, Influence of peanut oil on, 94
——, ——, Lymphocytes and, 84

Bacilli, Tubercle, on nonprotein medium, 93
——, ——, Virulence of, 93
Bacillus, tubercle, avian, Sensitization of cattle by, 9
——, ——, Morphological variation of, 95
——, vole, Vaccination of guinea pigs with, 103
Bacteria, acid-fast, Receptor analysis of, 105
—— in tuberculous cavities, 8
Bacterium, Acid-fast, in swine, 10
Bailey, R. L., Jr., and Stewart, H. J. Pneumothorax and circulation, 91
Bair, G., and Fineman, A. H. Tuberculin patch test, 106
Baker, A. B. See Diehl, H. S., *et al.*, 142
Balbi Robecco, J. M. Selective collapse, 114
Bang's disease, Tuberculin reaction and, 11
Barach, A. L., Martin, J., and Eckman, M. Acute pulmonary oedema, 152
Baur, O. Meningococci and tubercle bacilli in spinal fluid, 81
BCG in vaseline oil, 85
——, Sensitizing guinea pigs to, 104
—— vaccination, 105
Beach, B. A. See Hastings, E. G., *et al.*, 11
——, ——. ——. See McCarter, Janet R., *et al.*, 9
Beal, J. R. Pleural effusions, 65
——, ——. ——. Tuberculosis in adolescent females, 48
Bellicchi, O. M. Empyema treated with prontosil, 66
Bence, A. E. Sputum examination of each lung, 98
Benjamin, J. E., and Landt, H. Rebreathing experiments, 89
Benjamin, P. V., and Verghese, M. C. Diabetes and tuberculosis, 60
Bennett, G. See Sweet, P., *et al.*, 76
Berkelhamer, L. H. Settling characteristics of dusts, 161
Bertelli, J. A., and Vivoli, D. Tuberculosis of tonsils, 69
Bett, C., and Enquin, B. X-ray study of apical tuberculosis, 108
Bilateral plombage, 2
Bivings, L. Asthmatic bronchitis in children, 141
Blackford, S. D., and Casey, C. J. Tularemia of lung, 131
Bladder, Tuberculosis of, 79
Blades, B., and Myers, I. W. Bronchography in suppurative and neoplastic disease, 140
Bloch, R., and Bogardus, G. Cancer of lung, 18
——, ——., Tucker, W. B., and Bryant, J. E. X-ray survey in Negroes, 50
Bocchetti, F. Suction drainage of cavities, 5
Bogardus, G., and Bloch, R. Cancer of lung, 18
Bogart, F. B. Diagnosis of lung diseases, 106
Bohm, F. Pleural effusion, 64
Bone and joint tuberculosis, 71
—— tuberculosis, Early diagnosis of, 9
Bones and lung, Tuberculosis of the, 69
——, Cystic tuberculosis of, 72
——, Tuberculosis of, 71
Bosworth, D., and Haines, K. Spinal fusion, 73
Bottari, G., and Babolini, G. Intracavitary drainage, 118
——, ——. See Monaldi, V., *et al.*, 120
Bovine tubercle bacilli, Isolation of, 100
Bower, C. See Corper, H. J., *et al.*, 93
Boynton, R. E. See Myers, J. A., *et al.*, 47
Bracco, A. N., and Cetrangolo, A. A. Perforation of pleura in thoracoplasty, 118
Brailey, Miriam. Mortality in children of tuberculous households, 54
Braillon, J. See Douady, D., *et al.*, 37
Breathing, Dust particles removed by, 27
Bretey, J. Sensitizing guinea pigs to BCG, 104
——, ——. See Nègre, L., *et al.*, 93
Breton, A. Sarcoidosis, 82
Britton, J. A. See Sutton, F. C., *et al.*, 91
Bronchi and bronchioles, Histology of, 82
Bronchiectasis, 126
——, Congenital, 16
——, Cystic, 127
—— in tuberculosis of infancy, 53
Bronchioles, Histology of bronchi and, 82
Bronchitis, 16
——, Asthmatic, in children, 141
——, Infectious-allergic, 16
Bronchogenic carcinoma, 143, 144, 145, 146
—— ——, Bronchographic examination of, 147

Bronchogenic carcinoma, Excavated, 146
—— —— of lung in Chinese, 146
—— ——, Roentgen therapy for, 147
Bronchographic examination of bronchogenic carcinoma, 147
Bronchography, 109
—— in suppurative and neoplastic disease, 140
Bronchomoniliasis, 128
Bronchopleural fistula, 110
Bronchopneumonia, Atypical, 135, 137
—— of unknown aetiology, 133
Bronchospasm, Circulatory disturbances in, 90
Bronchostenosis, 149
Brooke, W. S., and Wells, A. Q. Vaccination of guinea pigs with vole bacillus, 103
Brucella infection, Hodgkin's disease and, 162
Brucellosis and tuberculosis, 34
Brügger, H. Cyst of lung, 151
Bryant, J. E. See Bloch, R. G., *et al.*, 50
Bryce, A. G. Lung abscess, 11
Bulnes, C., and Roma, F. Intracavitary drainage, 122
Büngeler, W., and Fernández, J. M. Leprolin, 163
Bunney, W. E., and Gottschall, R. Y. Skin reactions in guinea pigs, 85
Burnaud, R., and Francken, W. Suction drainage of cavities, 4
Buzzard, E. M. Thrombosis of superior vena cava, 40

Caffey, J. Obstructive emphysema, 149
Calderón Paul, E. Actinomycosis, 128
—— ——, ——. Tuberculous arthritis, 73
Campbell, J. A. Influenza and lung tumors in mice, 142
Cancer of lung, 17, 18, 19, 21
—— —— —— and subpleural scars, 20
—— —— ——, Metastases of, 21
Capacity, Cardiopulmonary, 91
Carcinoma, Bronchogenic, 143, 144, 145, 146
——, ——, Bronchographic examination of, 147
——, ——, Excavated, 146
——, ——, of lung in Chinese, 146
——, ——, Roentgen therapy for, 147
——, Pneumonectomy for, 21
Cardiopulmonary capacity, 91
Carr, J. G. See Sutton, F. C., *et al.*, 91
Carter, R. A. Overexpansion of lung in infancy, 109
Casey, C. J., and Blackford, S. D. Tularemia of lung, 131
Castelli, A. Treatment of empyema, 2
Castillo, P. A., and Jimenez, J. A. Metastases of cancer of lung, 21
Castleman, B., and Hampton, A. O. Pulmonary embolism, 151
Cattle, Sensitization of, by avian tubercle bacillus, 9
Cavernoscopy, 124
Cavities, 56
—— and collapse therapy, 112
—— after thoracoplasty, 117
——, Suction drainage of, 3, 4, 5, 6, 7
——, tuberculous, Bacteria in, 8
Celaya, M., and Olguin, V. Chronic pneumonitis, 13
Cellan-Jones, C. J., and Murphy, W. Chylothorax following trauma, 159
Cenci, V. P. See Phelps, P. S., *et al.*, 52
Cetrangolo, A. A., and Bracco, A. N. Perforation of pleura in thoracoplasty, 118
Chandler, F. A., and Page, M. A. Tuberculosis of spine, 74
Chang, F. C. See Hsieh, C. K., *et al.*, 146
Changes, Electrolyte, in tuberculosis of lung, 92
Chemotropism, 96
Chest injuries, 159
—— roentgenograms in pregnancy, 49
——, strapping of, Oedema of legs following, 153
Chiari, O. Tuberculosis in adolescents, 48
Childhood tuberculosis, 53
—— —— of lungs, 52
Children, Asthmatic bronchitis in, 141
——, Chinese, Tuberculosis in, 53
—— of tuberculous households, Mortality in, 54
——, Tuberculosis in, 52
Chinese, Bronchogenic carcinoma of lung in, 146
—— children, Tuberculosis in, 53
——, Tuberculosis of lung in, 51
Ch'iu, P. T. Y., Ho, E. A., and Li, C. C. Tuberculosis of lung in Chinese, 51
——, ——. ——. ——. See Myers, J. A., *et al.*, 47

Cholesterol granulomatosis of the lung, 32
Chronic miliary tuberculosis, 59
—— pneumonitis, 13
Chylothorax following trauma, 159
Circulation, Pneumothorax and, 91
——, pulmonary, Velocity of, 92
Circulatory disturbances in bronchospasm, 90
Clarke, B. R., and Erskine, S. L. W. Pneumothorax, 114
Climate and disease, 56
Coccidioides, Erythema nodosum in, 40
Coccidioidomycosis, Erythema nodosum in, 39
Cohen, A. G., and Kissin, M. Tuberculoma of lung, 59
Cohen, W., and Nathanson, L. Bone and joint tuberculosis, 71
——, ——. See Epstein, I. G., *et al.*, 109
Cohn, M. L., and Corper, H. J. Histaminase and tuberculo-allergy, 83
——, ——. ——. See Corper, H. J., *et al.*, 93
Cold vaccines, 142
Cole, G. C. Lungs in drug addicts, 33
Cole, L. G., and Cole, W. G. Silicosis, 22
Cole, W. G., and Cole, L. G. Silicosis, 22
Collapse, Selective, 114
—— therapy, Cavities and, 112
—— ——, Spirometry in, 90
Collins, R. J., and Macpherson, L. Tuberculosis of the bones and lung, 69
Colon, Interposition of, 1
Complications, Pulmonary, of renal suppuration, 141
Compounds, Oxidizing, and experimental tuberculosis, 83
Congenital absence of hemidiaphragm, 38
—— bronchiectasis, 16
Conjugal tuberculosis, 47
Conjunctival tuberculosis, 80
Conjunctivitis, Phlyctenular, 81
Control of tuberculosis in India, 51
——, Tuberculosis, 50
Corper, H. J., and Cohn, M. L. Histaminase and tuberculo-allergy, 83
——, ——. ——., Cohn, M. L., and Bower, C. Tubercle bacilli on nonprotein medium, 93
Costantini, G. Suction drainage of cavities, 6
Coulthard, H. S., and Harris, R. I. Tuberculosis of spine, 75
Cournand, A., Darling, R. C., Mansfield, J. S., and Richards, D. W., Jr. Residual air, 87
——, ——. See Darling, R. C., *et al.*, 87, 88
Cowan, D. W. See Diehl, H. S., *et al.*, 142
Craige, B., Jr. Status asthmaticus, 157
Craver, L. F. Bronchogenic carcinoma, 144
——, ——. ——. Malignant tumors of lung, 142
Crushing, Phrenic, 1
Cruz Muñoz, L. A., and Sanchez Acosta, R. Tuberculosis of lung and intestine, 58
Cuba, Silico-tuberculosis in, 63
Cultural diagnosis of tuberculosis, 97
Culturing tubercle bacilli from sputum, 98
Cummins, S. L. Anthracite and tubercle bacilli, 96
Curtzwiler, F. C., and Peirce, C. B. Tuberculous tracheobronchitis, 67
Cyst, Hydatid, 28
—— of lung, 150, 151
Cystic bronchiectasis, 127
—— tuberculosis of bones, 72
Cysts, Lung, 28

Damage, X-ray, to lung, 31
Darling, R. C., Cournand, A., and Richards, D. W., Jr. Residual air, 88
——, ——. ——., ——, ——., Mansfield, J. S., and Richards, D. W., Jr. Nitrogen excretion, 87
——, ——. ——. See Cournand, A., *et al.*, 87
Darricarrere, T. See Orrego, H., *et al.*, 54
Davidson, L. R. Intracavitary drainage, 124
Davies, T. W. Silicosis in slate quarriers, 25
Dayman, H. See Thorn, G. W., *et al.*, 92
DeBakey, M., and Ochsner, A. Bronchogenic carcinoma, 143
Decline of tuberculosis, 43
Delahant, A. B. See Vorwald, A. J., *et al.*, 26
Density, Marginal, of lung in pneumothorax, 115
Desmeules, R. Asthma and tuberculosis, 61
Detergents, Antiseptics and, 97

Diabetes and pulmonary infection, 124
—— —— tuberculosis, 60
Diagnosis, Cultural, of tuberculosis, 97
——, Early, of bone tuberculosis, 9
—— of lung diseases, 106
—— —— tuberculosis, 108
Diaz Rivera, R. S. Bronchomoniliasis, 128
Dick, V. S., and Nesbit, R. M. Pulmonary complications of renal suppuration, 141
Diebold, O. Bilateral plombage, 2
Diehl, H. S., Baker, A. B., and Cowan, D. W. Cold vaccines, 142
——, ——. ——. See Myers, J. A., *et al.*, 47
Dietl, K., and Koszler, V. Avian tuberculin, 9
Disability, lung disease, Industrial, 160
Disease, Climate and, 56
——, suppurative and neoplastic, Bronchography in, 140
Diseases, lung, Diagnosis of, 106
Dog, Tuberculous septicaemia in, 83
Dold, H., and Ochsenreither, F. Protective function of saliva, 106
Doll, E. R., and Durant, A. J. Aspergillosis in a skunk, 17
Dominicis, F., and Martinez, V. Pulmonary actinomycosis, 16
Douady, D., Braillon, J., and Georges, G. Pneumococcic empyema, 37
Dougherty, T. F., and Turley, L. A. Lymphocytes and tubercle bacilli, 84
Douglas, B. H., and Tompkins, E. Silicotuberculosis, 27
Drainage, Intracavitary, 118, 120, 121, 122, 123, 124
——, ——, after thoracoplasty, 124
——, Suction, of cavities, 3, 4, 5, 6, 7
Drug addicts, Lungs in, 33
Durant, A. J., and Doll, E. R. Aspergillosis in a skunk, 17
Dust particles removed by breathing, 27
Dusts, Settling characteristics of, 161
Dworski, M. See Vorwald, A. J., *et al.*, 26

Early diagnosis of bone tuberculosis, 9
Ebina, T. Receptor analysis of acid-fast bacteria, 105
Eckman, M. See Barach, A. L., *et al.*, 152
Effects of industrial gases upon lung, 35
Effusion, Pleural, 64
Effusions, Pleural, 65
Ehler, A. A., and Maier, H. C. Oesophagus after pneumonectomy, 2
Ehrlich, D. E., and Robins, A. B. Group X-ray surveys, 49
Electrolyte changes in tuberculosis of lung, 92
Ellis, R., and Hirsh, B. Oedema of legs following strapping of chest, 153
Embolism, Pulmonary, 151
Emphysema, 148
——, Interstitial, 30
——, —— and mediastinal, 149
——, Obstructive, 148, 149
——, pulmonary, Atherosclerosis of pulmonary artery and, 29
Employee with tuberculosis, 46
Empyema, 65
——, Pneumococcic, 37
—— treated with prontosil, 66
——, Treatment of, 2
——, Tuberculous, 66
——, Unexpandable lung in, 154
Endometritis, Tuberculous, 78
Endometrium, Tuberculosis of, 78
Engelberg, H. See Freiman, D., *et al.*, 34
Engelstad, R. B. Radiation reaction in lung, 155
Enquin, B., and Bett, C. X-ray study of apical tuberculosis, 108
——, ——. See Vaccarezza, R. F., *et al.*, 46
Epidemiology of tuberculosis, 45
Epididymitis, Tuberculous, 78
Epituberculosis, 53
Epstein, I. G., Herman, M., Green, H., and Cohen, W. Tomography, 109
Erdstein, S. F. See Vaccarezza, R. F., *et al.*, 86
Erskine, S. L. W., and Clarke, B. R. Pneumothorax, 114
Erythema nodosum in coccidioides, 40
—— —— —— coccidioidomycosis, 39
Espinosa Nodal, G. A. Cavities, 56
Evans, E. E. Effects of industrial gases upon lung, 35
Examination, Bronchographic, of bronchogenic carcinoma, 147
—— of faeces in tuberculosis, 98
Excavated bronchogenic carcinoma, 146
Excretion, Nitrogen, 87
Experimental silicosis and pneumonia, 26
—— tuberculosis, Oxidizing compounds and, 83

Experiments, Rebreathing, 89
Extracts from leprous spleens, Skin tests with, 163
Extrapleural pneumothorax, 115
Extrapulmonary tuberculosis, 64
Exudates of subphrenic origin, 141
Eye, tuberculosis of, Treatment of, 80

Faeces, Examination of, in tuberculosis, 98
Fanano, V. Congenital absence of hemidiaphragm, 38
Farinas, P. L. Bronchographic examination of bronchogenic carcinoma, 147
Feinberg, S. M. See Rubin, S. S., *et al.*, 38
Feldman, H. A., and Hanks, J. H. Tubercle bacilli from spinal fluids, 100
——, ——. ——., —— ——, ——. ——. Tubercle bacilli in urine, 99
Feldman, W. H., and Karlson, A. G. Acid-fast bacterium in swine, 10
——, ——. ——., —— ——, ——. ——. Tubercle bacilli in swine tonsils, 10
Females, adolescent, Tuberculosis in, 48
Fernández, J. M., and Büngeler, W. Leprolin, 163
Ferroni, P. Tuberculous septicaemia in dog, 83
Fetter, J. S. See Widmann, B. P., *et al.*, 68
Fibrin body, 115
Fibrosis, Massive, in obstructive tracheobronchial tuberculosis, 60
—— of lung in scleroderma, 161
Films, Miniature, 107
——, X-ray, Miniature, 107
Fineberg, S. M., Foran, F. L., Lichtenstein, M. R., Padnos, E., Rappaport, B. Z., Sheldon, J., and Zeller, M. Oral pollen therapy, 158
Fineman, A. H., and Bair, G. Tuberculin patch test, 106
Finney, G. G., and Fisher, A. M. Lung abscess, 12
Fischer, Hanna. Childhood tuberculosis, 53
Fisher, A. M., and Finney, G. G. Lung abscess, 12
Fisher, J. H., and Macklin, C. C. Interstitial and mediastinal emphysema, 149
Fissure, Transverse interlobar, 58
Fistula, Bronchopleural, 110
Fluoroscopic roentgenography, 109
Fojanini, G., and Zorzoli, G. Bacteria in tuberculous cavities, 8
Follow-up in tuberculosis, 112
Foran, F. L. See Fineberg, S. M., *et al.*, 158
Francken, W., and Burnaud, R. Suction drainage of cavities, 4
Frank, L. W. Phrenic crushing, 1
Franklin, R. M. Lung abscess, 11
Freiman, D., Engelberg, H., and Merrit, W. Oil pneumonia, 34
Freund, R., and Samuelson, S. Löffler's syndrome, 132
Fried, J. R., and Goldberg, H. Radiation reaction in lung, 155
Friedländer lung infections, 130
—— pneumonia, 130
Friedrich, G. Cancer of lung and subpleural scars, 20
Frimodt-Möller, C. Control of tuberculosis in India, 51
Fripp, A. T. Early diagnosis of bone tuberculosis, 9
Fuller's earth, Pneumonoconiosis from, 160
Function, Protective, of saliva, 106
——, Respiratory, in tuberculosis, 89
Fusion, Spinal, 73

Gaerber, H., and Kunz, H. Results of thoracoplasty, 117
Gallone, L. Chronic miliary tuberculosis, 59
Garcia Cerviño, R. Brucellosis and tuberculosis, 34
García Laredo, J. E. Transient pulmonary opacities, 133
García Suárez, E. See Orrego, H., *et al.*, 54
Gases, industrial, Effects of, upon lung, 35
Geberding, H. Primary tuberculosis in adults, 54
Georges, G. See Douady, D., *et al.*, 37
Gerson, M. J. See Murphy, J. R., *et al.*, 161
Goldberg, H., and Fried, J. R. Radiation reaction in lung, 155
Golden, R. Obstructive emphysema, 148
Goldman, L. B. Hodgkin's disease, 39
Gosselin, J. Bronchography, 109
Gottschall, R. Y., and Bunney, W. E. Skin reactions in guinea pigs, 85

Graham, H. K. Chest roentgenograms in pregnancy, 49
Granulomatosis, Cholesterol, of the lung, 32
Grass, H. Fluoroscopic roentgenography, 109
——, ——. Suction drainage of cavities, 7
Green, H. See Epstein, I. G., *et al.*, 109
——, ——. See Leon, J. L., *et al.*, 117
Greenberger, A. J., Greenberger, M. E., and Rosenthal, M. Ureterostomy, 79
Greenberger, M. E., and Helfert, I. Tuberculosis of bladder, 79
——, ——. ——. See Greenberger, A. J., *et al.*, 79
Greenfield, J. Bronchiectasis, 126
Group X-ray surveys, 49
Growth of tubercle bacilli, Inhibition of, 97
Guinea pigs, Sensitizing, to BCG, 104
—— ——, Skin reactions in, 85
—— ——, Vaccination of, with vole bacillus, 103

Haas, S. L. Tuberculosis of knee, 77
Haight, C., and Alexander, J. Hydatid cyst, 28
Haines, K., and Bosworth, D. Spinal fusion, 73
Halpert, B. Bronchogenic carcinoma, 143
Halton, J. Chest injuries, 159
Hamman, L. Spontaneous pneumothorax, 36
Hampton, A. O., and Castleman, B. Pulmonary embolism, 151
Hanks, J. H., and Feldman, H. A. Tubercle bacilli from spinal fluids, 100
——, ——. ——., —— ——, ——. ——. Tubercle bacilli in urine, 99
Harrington, F. E. See Myers, J. A., *et al.*, 45
Harris, R. I., and Coulthard, H. S. Tuberculosis of spine, 75
Hartman, M. M. Friedländer pneumonia, 130
Hasler, J. Congenital bronchiectasis, 16
Hastings, E. G., McCarter, Janet R., Beach, B. A., Wisnicky, W., and Healy, J. S. Tuberculin reaction and Bang's disease, 11
——, ——. ——. See McCarter, Janet R., *et al.*, 9
Hauser, H., and Wolpaw, S. E. Excavated bronchogenic carcinoma, 146
Havens, W. P., and Reimann, H. A. Pneumonia, 14
Haviland, J. W., and Long, P. H. Treatment of pneumonia, 139
Hay fever, Histaminase in, 38
—— ——, Potassium in, 38, 39
Healed miliary tubercles, 82
Healy, J. S. See Hastings, E. G., *et al.*, 11
Heart in tuberculosis, 63
Helfert, I., and Greenberger, M. E. Tuberculosis of bladder, 79
Hemidiaphragm, Congenital absence of, 38
Herholz, G. Pneumonolysis, 116
Herman, M., and Palitz, L. Tuberculous empyema, 66
——, ——. See Epstein, I. G., *et al.*, 109
Herniation, Mediastinal, 111
Hirsch, I. S. Miniature X-ray films, 107
Hirsh, B., and Ellis, R. Oedema of legs following strapping of chest, 153
Histaminase and tuberculo-allergy, 83
—— in hay fever, 38
Histology of bronchi and bronchioles, 82
Ho, E. A. See Ch'iu, P. T. Y., *et al.*, 51
Hochberg, L. A. Unexpandable lung in empyema, 154
——, ——. ——. See Rabinovitch, J., *et al.*, 21
Hodgkin's disease, 39
—— —— and Brucella infection, 162
Holinger, P., and Radner, D. B. Bronchogenic carcinoma, 145
Holman, E. F., and Samson, P. C. Pneumonectomy for carcinoma, 21
Hosmer, Elizabeth. Perihilar shadows, 58
Households, tuberculous, children of, Mortality in, 54
Howard, P. J. Phlyctenular conjunctivitis, 81
Howard, R. P. See Thorn, G. W., *et al.*, 92
Hsieh, C. K., Wang, S. H., and Chang, F. C. Bronchogenic carcinoma of lung in Chinese, 146
Hsu, C.-L., and T'ang, B. H. Y. Transverse interlobar fissure, 58
Hughes, J. G., and Simpson, W. L. Bronchiectasis in tuberculosis of infancy, 53

Hunter, A. F., and Jenkinson, E. L. Cancer of lung, 18
Hydatid cyst, 28

Idiopathic pneumothorax, 36
Index of activity, 111
India, Control of tuberculosis in, 51
Industrial gases, Effects of, upon lung, 35
—— lung disease disability, 160
Industrialization, Tuberculosis and, 45
Infancy, Overexpansion of lung in, 109
——, tuberculosis of, Bronchiectasis in, 53
Infection, pulmonary, Diabetes and, 124
——, respiratory, Acute, 138
Infections, lung, Friedländer, 130
Infectious-allergic bronchitis, 16
Infiltrations, lung, Transitory, 11
Influence of peanut oil on tubercle bacilli, 94
Influenza and lung tumors in mice, 142
Ingraham, E. S., and Wartman, W. B. Chemotropism, 96
Inhibition of growth of tubercle bacilli, 97
Injuries, Chest, 159
Intercostal nerves, Temporary paralysis of, 2
Interposition of colon, 1
Interstitial and mediastinal emphysema, 149
—— emphysema, 30
Intestine, Tuberculosis of lung and, 58
Intracavitary drainage, 118, 120, 121, 122, 123, 124
—— —— after thoracoplasty, 124
Isolation of bovine tubercle bacilli, 100

Jacobsen, V. C. Radiation changes in lungs, 155
Jagić, N. Circulatory disturbances in bronchospasm, 90
Jaime, O. Tubercle bacilli in laryngeal content, 98
Jarisch, A., Richter, H., and Thoma, H. Pulmonary oedema, 37
Jenkinson, E. L., and Hunter, A. F. Cancer of lung, 18
Jensen, R. M., and McDonald, J. R. Tuberculosis of endometrium, 78
Jimenez, J. A., and Castillo, P. A. Metastases of cancer of lung, 21
Jimenez, M. Intracavitary drainage, 123
Joint tuberculosis, Bone and, 71
Jordan, L. S. Tuberculosis control, 50

Kaplan, M. A. See Rubin, S. S., *et al.*, 38
Karlson, A. G. Isolation of bovine tubercle bacilli, 100
——, ——. ——., and Feldman, W. H. Acid-fast bacterium in swine, 10
——, ——. ——., —— ——, ——. ——. Tubercle bacilli in swine tonsils, 10
Kartagener, M., and Ulrich, K. Tracheobronchiectasis, 127
Katz, H. L. Aortic aneurysm, 162
Keeney, E. L. Histaminase in hay fever, 38
Kelly, C. H. Bronchogenic carcinoma, 145
Key, L. A. Tuberculosis of knee, 77
Kicklin, B. R., and Olds, J. W. Bronchogenic carcinoma, 146
King, E. S., and Lucas, M. Culturing tubercle bacilli from sputum, 98
Kinghorn, H. M. Diagnosis of tuberculosis, 108
Kirklin, B. R. Lipoid pneumonia, 154
Kissin, M., and Cohen, A. G. Tuberculoma of lung, 59
Knee, Tuberculosis of, 77
Kneeland, Y., Jr., and Smetana, H. F. Atypical bronchopneumonia, 137
Knies, P. T. Marginal density of lung in pneumothorax, 115
Kornblum, K., and Reimann, H. A. Acute respiratory infection, 138
Koszler, V., and Dietl, K. Avian tuberculin, 9
Krainin, P. See Murphy, J. R., *et al.*, 161
Kremer, W. Intracavitary drainage, 121
Krombach, E., and Schairer, E. X-ray damage to lung, 31
Krusen, F. H. Ultraviolet radiation, 112
Kunz, H., and Gaerber, H. Results of thoracoplasty, 117

Lackner, J. E., Schiller, W., and Tulsky, A. S. Tuberculous endometritis, 78
Landt, H., and Benjamin, J. E. Rebreathing experiments, 89
Laporte, R. Allergy-producing properties of paratubercle bacilli, 101
Laryngeal content, Tubercle bacilli in, 98
Larynx, Tuberculosis of, 67
——, —— —— lung and, 58
Lavaux, R. See Nègre, L., *et al.*, 93

Law, D. E., and Schaefer, G. Pregnancy in tuberculosis, 62
Law, J. J. Cystic tuberculosis of bones, 72
Lawson, Alice. See Phelps, P. S., *et al.*, 52
Leddy, E. T., and Moersch, H. J. Roentgen therapy for bronchogenic carcinoma, 147
Lederer, M. See Rabinovitch, J., *et al.*, 21
Legs, Oedema of, following strapping of chest, 153
Lehmann, A. H., and Schwartz, S. F. Pelvic tuberculosis, 78
Leon, J. L., Green, H., and Serbst, C. A. Cavities after thoracoplasty, 117
Leprolin, 163
Leprous spleens, extracts from, Skin tests with, 163
Leucocyte count in tuberculosis, 86
Li, C. C. See Ch'iu, P. T. Y., *et al.*, 51
Lichtenstein, M. R. See Fineberg, S. M., *et al.*, 158
Liège, R. Tonsillectomy and tuberculosis, 54
Lincoln, Edith M. Tuberculosis in children, 52
Lipoid pneumonia, 154
Ljunggren, E. Tuberculous epididymitis, 78
Löffler's syndrome, 132
Long, E. R. Decline of tuberculosis, 43
Long, P. H., and Haviland, J. W. Treatment of pneumonia, 139
Longcope, W. T. Bronchopneumonia of unknown aetiology, 133
Lorge, H. J. Idiopathic pneumothorax, 36
Lower lobe tuberculosis, 55
Lucas, M., and King, E. S. Culturing tubercle bacilli from sputum, 98
Lues, Pulmonary, 15
Lung abscess, 11, 12, 125
—— and intestine, Tuberculosis of, 58
—— —— larynx, Tuberculosis of, 58
——, Bronchogenic carcinoma of, in Chinese, 146
——, Cancer of, 17, 18, 19, 21
——, —— ——, and subpleural scars, 20
——, —— ——, Metastases of, 21
——, Cholesterol granulomatosis of the, 32
——, Cyst of, 150, 151
—— cysts, 28
—— disease disability, Industrial, 160
—— diseases, Diagnosis of, 106
Lung, Effects of industrial gases upon, 35
——, Fibrosis of, in scleroderma, 161
—— infections, Friedländer, 130
—— infiltrations, Transitory, 11
——, Malignant tumors of, 142
——, Marginal density of, in pneumothorax, 115
——, Nonputrid suppuration of, 125
——, Overexpansion of, in infancy, 109
——, Phagocytosis in, 101
——, Radiation reaction in, 155, 156
——, Sarcoidosis of, 63
——, Sputum examination of each, 98
——, Syphilis of, 129
——, Tuberculoma of, 59
——, Tuberculosis of, 58
——, —— ——, Electrolyte changes in, 92
——, —— ——, in Chinese, 51
——, —— —— the bones and, 69
——, —— ——, Treatment of, 113
——, Tularemia of, 131
—— tumors, Influenza and, in mice, 142
——, —— of, Tomography in, 147
——, Unexpandable, in empyema, 154
——, X-ray damage to, 31
Lungs, Childhood tuberculosis of, 52
—— in drug addicts, 33
——, Radiation changes in, 155
Lupus vulgaris, Treatment of, 81
Lymph node tuberculosis, 68
Lymphocytes and tubercle bacilli, 84

MacIntosh, C. A. Extrapleural pneumothorax, 115
Macklin, C. C. Interstitial emphysema, 30
——, ——. ——., and Fisher, J. H. Interstitial and mediastinal emphysema, 149
Macpherson, L., and Collins, R. J. Tuberculosis of the bones and lung, 69
Maier, H. C. Mediastinal herniation, 111
——, ——. ——., and Ehler, A. A. Oesophagus after pneumonectomy, 2
Malbrán, J. See Roberto Paso, J., *et al.*, 80
Malignant tumors of lung, 142
Mansfield, J. S. See Cournand, A., *et al.*, 87
——, ——. ——. See Darling, R. C., *et al.*, 87

Mapes, R., and Rothman, P. E. Massive fibrosis in obstructive tracheobronchial tuberculosis, 60
Marginal density of lung in pneumothorax, 115
Mariono Varona, A. Tuberculosis of larynx, 67
Martens, G. Transitory lung infiltrations, 11
Martin, J. See Barach, A. L., *et al.*, 152
Martinez, V. Syphilis and tuberculosis, 62
——, ——., and Dominicis, F. Pulmonary actinomycosis, 16
Martos Vallhonrat, J. Syphilis of lung, 129
Massive fibrosis in obstructive tracheobronchial tuberculosis, 60
Matson, R. C. Treatment of tuberculosis of lung, 113
Matsuzawa, D. Bronchopleural fistula, 110
——, ——., and Samuels, H. Pleural effusions, 65
Mauderli, H. Tuberculosis in army, 49
Mayer, E. Industrial lung disease disability, 160
Mayer, L. Tuberculosis of spine, 74
McCarter, Janet R., Hastings, E. G., and Beach, B. A. Sensitization of cattle by avian tubercle bacillus, 9
——, —— ——. See Hastings, E. G., *et al.*, 11
McCracken, J. P., and Allan, W. B. Aneurysm of pulmonary artery, 162
McDonald, J. R., and Jensen, R. M. Tuberculosis of endometrium, 78
McNally, W. D. Acute silicosis, 160
——, ——. ——., and Trostler, I. S. Pneumonoconiosis from Fuller's earth, 160
Mean, H., and Rossier, P. H. Respiratory function in tuberculosis, 89
Mediastinal emphysema, Interstitial and, 149
—— herniation, 111
Medium, nonprotein, Tubercle bacilli on, 93
Meisels, E. Sarcoidosis of lung, 63
Meningococci and tubercle bacilli in spinal fluid, 81
Merrit, W. See Freiman, D., *et al.*, 34
Metastases of cancer of lung, 21
Mice, Influenza and lung tumors in, 142
Miliary tubercles, Healed, 82
—— tuberculosis, Chronic, 59
Miller, H., and Piness, G. Potassium in hay fever, 39
Miniature films, 107
—— X-ray films, 107
Mishulow, L., and Reavin, S. Cultural diagnosis of tuberculosis, 97
Moersch, H. J., and Leddy, E. T. Roentgen therapy for bronchogenic carcinoma, 147
——, ——. ——., —— Prickman, L. E. Bronchostenosis, 149
Monaldi, V. Intracavitary drainage, 121
——, ——. Suction drainage of cavities, 5
——, ——., Bottari, G., and Babolini, G. Intracavitary drainage, 120
Monod, O. Pneumonia, 14
Moolten, S. E. Diabetes and pulmonary infection, 124
Moore, S. Tomography in tumors of lung, 147
Morelli, E. Suction drainage of cavities, 3
Morera Carbonell, J. Syphilis of lung, 129
Morphological variation of tubercle bacillus, 95
Morse, H. D., and Scott, D. L. Tuberculosis of kidney, 79
Mortality in children of tuberculous households, 54
Müller, R. W., and Argemi, J. Pericavitary atelectasis, 4
Murphy, J. R., Krainin, P., and Gerson, M. J. Fibrosis of lung in scleroderma, 161
Murphy, W., and Cellan-Jones, C. J. Chylothorax following trauma, 159
Murray, M. E. Atypical bronchopneumonia, 135
Muschenheim, C., and Amberson, J. B., Jr. Interposition of colon, 1
Myers, I. W., and Blades, B. Bronchography in suppurative and neoplastic disease, 140
Myers, J. A., Boynton, R. E., Diehl, H. S., Streukens, T. L., and Ch'iu, P. T. Y. Tuberculosis in nurses, 47
——, ——. ——., Harrington, F. E., Sprague, E., and Pérez, J. A. Epidemiology of tuberculosis, 45

Naeslund, C. Prevention of silicosis, 25
Nathanson, L., and Cohen, W. Bone and joint tuberculosis, 71
Navarrete Sierra, A. Silico-tuberculosis in Cuba, 63
Nègre, L., Bretey, J., Lavaux, R., and Simonet, Mlle. Virulence of tubercle bacilli, 93
Negroes, X-ray survey in, 50
Neoplastic disease, suppurative and, Bronchography in, 140
Nesbit, R. M., and Dick, V. S. Pulmonary complications of renal suppuration, 141
Neuber, E. Treatment of lupus vulgaris, 81
Neumann, W. Index of activity, 111
——, ——. Tuberculosis and asthma, 60
Niederland, W. Tuberculosis and industrialization, 45
Nitrogen excretion, 87
Nonputrid suppuration of lung, 125
Nordgren, B. O. Phrenic paralysis, 1
Nurses, Tuberculosis in, 47

Obstructive emphysema, 148, 149
—— tracheobronchial tuberculosis, Massive fibrosis in, 60
Ochsenreither, F., and Dold, H. Protective function of saliva, 106
Ochsner, A., and DeBakey, M. Bronchogenic carcinoma, 143
Oedema of legs following strapping of chest, 153
——, Pulmonary, 37
——, ——, Acute, 152
Oesophagus after pneumonectomy, 2
Oil pneumonia, 34
Olds, J. W., and Kicklin, B. R. Bronchogenic carcinoma, 146
Olguin, V., and Celaya, M. Chronic pneumonitis, 13
Oobayashi, Y. See Yanagisawa, K., *et al.*, 83
Opacities, pulmonary, Transient, 133
Oral pollen therapy, 158
Orrego, H., García Suárez, E., and Darricarrere, T. Primary tuberculosis in adults, 54
Ostrum, H. W. See Widmann, B. P., *et al.* 68
Oswald, N. C. Tubercle bacilli in rats, 84
Overexpansion of lung in infancy, 109
Overholt, R. H. Pneumonectomy, 2
——, ——. ——., and Rumel, W. R. Cancer of lung, 19
Oxidizing compounds and experimental tuberculosis, 83

Padnos, E. See Fineberg, S. M., *et al.*, 158
Page, M. A., and Chandler, F. A. Tuberculosis of spine, 74
Palitz, L., and Herman, M. Tuberculous empyema, 66
Paralysis, Phrenic, 1
——, Temporary, of intercostal nerves, 2
Paratubercle bacilli, Allergy-producing properties of, 101
Parker, R. L. Emphysema, 148
Parodi, F. Intracavitary drainage, 120
Passalacqua, L. A. Atelectasis, 32
Patch test, Tuberculin, 106
Paterson, J. F. Conjugal tuberculosis, 47
Patterson, H. S., and van Wijk, A. M. Dust particles removed by breathing, 27
Peanut oil, Influence of, on tubercle bacilli, 94
Pearlman, A. W. Amyloidosis, 163
Peirce, C. B., and Curtzwiler, F. C. Tuberculous tracheobronchitis, 67
Pelvic tuberculosis, 78
Penetrating wounds of thorax, 34
Pérez, J. A. See Myers, J. A., *et al.*, 45
Perforation of pleura in thoracoplasty, 118
Pericavitary atelectasis, 4
Perihilar shadows, 58
Petroff, S. A., and Schain, P. Antiseptics and detergents, 97
——, ——. ——., —— ——, ——. Inhibition of growth of tubercle bacilli, 97
Phagocytosis in lung, 101
Phelps, P. S., Cenci, V. P., and Lawson, Alice. Tuberculin survey, 52
Phlyctenular conjunctivitis, 81
Phrenic crushing, 1
—— paralysis, 1
Piness, G., and Miller, H. Potassium in hay fever, 39
Pisaecka-Zeyland, J., and Zeyland, J. BCG vaccination, 105
Pleura, Perforation of, in thoracoplasty, 118

Pleural effusion, 64
—— effusions, 65
Plombage, Bilateral, 2
Pneumococcic empyema, 37
Pneumonectomy, 2
—— for carcinoma, 21
——, Oesophagus after, 2
Pneumonia, 14
——, Experimental silicosis and, 26
——, Friedländer, 130
——, Lipoid, 154
——, Oil, 34
——, Sulfapyridine in, 139
——, Treatment of, 139
Pneumonitis, Chronic, 13
Pneumonoconiosis from Fuller's earth, 160
Pneumonolysis, 116
Pneumothorax, 114
—— and circulation, 91
——, Extrapleural, 115
——, Idiopathic, 36
——, Marginal density of lung in, 115
——, Spontaneous, 36
Pohle, E. A., and Ritchie, G. Silicosis and tuberculosis, 111
Pollen therapy, Oral, 158
Poston, M. A., and Wise, N. B. Hodgkin's disease and Brucella infection, 162
Potassium in hay fever, 38, 39
Pottenger, F. M. Climate and disease, 56
Potter, M. G. Pregnancy and tuberculosis, 61
Pregnancy and tuberculosis, 61
——, Chest roentgenograms in, 49
—— in tuberculosis, 62
Prevention of silicosis, 25
Prickman, L. E., and Moersch, H. J. Bronchostenosis, 149
Primary tuberculosis in adults, 54, 55
Program, Tuberculosis, 51
Prontosil, Empyema treated with, 66
Protective function of saliva, 106
Pulmonary actinomycosis, 16
—— artery, Aneurysm of, 162
—— ——, Atherosclerosis of, and pulmonary emphysema, 29
—— circulation, Velocity of, 92
—— complications of renal suppuration, 141
—— embolism, 151
Pulmonary emphysema, Atherosclerosis of pulmonary artery and, 29
—— infection, Diabetes and, 124
—— lues, 15
—— oedema, 37
—— ——, Acute, 152
—— opacities, Transient, 133

Quarriers, slate, Silicosis in, 25

Rabinovitch, J., Hochberg, L. A., and Lederer, M. Cancer of lung, 21
Radiation changes in lungs, 155
—— reaction in lung, 155, 156
——, Ultraviolet, 112
Radner, D. B., and Holinger, P. Bronchogenic carcinoma, 145
Rappaport, B. Z. See Fineberg, S. M., *et al.*, 158
Rats, Tubercle bacilli in, 84
Reaction, Radiation, in lung, 155, 156
——, Tuberculin, and Bang's disease, 11
Reactions, Skin, in guinea pigs, 85
Reavin, S., and Mishulow, L. Cultural diagnosis of tuberculosis, 97
Rebreathing experiments, 89
Receptor analysis of acid-fast bacteria, 105
Regulation of respiration, 86
Reichle, H. S., and Work, J. L. Healed miliary tubercles, 82
Reid, Ada Chree. Annual surveys, 46
——, —— ——. Employee with tuberculosis, 46
——, —— ——. Tuberculosis of lung, 58
Reimann, H. A., and Havens, W. P. Pneumonia, 14
——, ——. ——., —— Kornblum, K. Acute respiratory infection, 138
Reisner, D., and Tchertkoff, J. G. Cystic bronchiectasis, 127
Renal suppuration, Pulmonary complications of, 141
Residual air, 87, 88
Respiration, Regulation of, 86
Respiratory allergy, 158
—— function in tuberculosis, 89
—— infection, Acute, 138
—— tetany, 31
Rest, A. Fibrin body, 115
Results of thoracoplasty, 117

Rettger, L. F., and Vera, H. D. Morphological variation of tubercle bacillus, 95
Rey, J. C. See Vaccarezza, R. F., *et al.*, 86
Richards, D. W., Jr. See Cournand, A., *et al.*, 87
——, ——. ——., ——. See Darling, R. C., *et al.*, 87, 88
Richter, H. See Jarisch, A., *et al.*, 37
Ritchie, G., and Pohle, E. A. Silicosis and tuberculosis, 111
Riva, A. See Roberto Paso, J., *et al.*, 80
Roberto Paso, J., Malbrán, J., and Riva, A. Conjunctival tuberculosis, 80
Robertson, O. H. Phagocytosis in lung, 101
Robins, A. B., and Ehrlich, D. E. Group X-ray surveys, 49
Rodriguez, E. J. See Vaccarezza, R. F., *et al.*, 46
Roentgen therapy for bronchogenic carcinoma, 147
Roentgenograms, Chest, in pregnancy, 49
Roentgenography, Fluoroscopic, 109
Roma, F., and Bulnes, C. Intracavitary drainage, 122
Rosenthal, M. See Greenberger, A. J., *et al.*, 79
Rossier, P. H. Respiratory tetany, 31
——, ——. ——., and Mean, H. Respiratory function in tuberculosis, 89
Rothman, P. E., and Mapes, R. Massive fibrosis in obstructive tracheobronchial tuberculosis, 60
Rubin, S. S., Aaronson, A. L., Kaplan, M. A., and Feinberg, S. M. Potassium in hay fever, 38
Rumel, W. R., and Overholt, R. H. Cancer of lung, 19

Saenz, A., and Urquijo, C. A. BCG in vaseline oil, 85
Sailer, S. Epituberculosis, 53
Saliva, Protective function of, 106
Samson, P. C., and Holman, E. F. Pneumonectomy for carcinoma, 21
Samuels, H., and Matsuzawa, D. Pleural effusions, 65
Samuelson, S., and Freund, R. Löffler's syndrome, 132
Sanchez Acosta, R., and Cruz Muñoz, L. A. Tuberculosis of lung and intestine, 58
Sante, L. R. Exudates of subphrenic origin, 141
Santi, L. R. Lung cysts, 28
Sarcoidosis, 82
—— of lung, 63
Scars, subpleural, Cancer of lung and, 20
Schaefer, G., and Law, D. E. Pregnancy in tuberculosis, 62
Schain, P., and Petroff, S. A. Antiseptics and detergents, 97
——, ——., —— ——, ——. ——. Inhibition of growth of tubercle bacilli, 97
Schairer, E., and Krombach, E. X-ray damage to lung, 31
Schiller, W. See Lackner, J. E., *et al.*, 78
Schmidt, P. G. Infectious-allergic bronchitis, 16
Schröder, G. Bronchitis, 16
Schuberth, A. Intracavitary drainage after thoracoplasty, 124
——, ——. Suction drainage of cavities, 4
Schwartz, S. F., and Lehmann, A. H. Pelvic tuberculosis, 78
Scleroderma, Fibrosis of lung in, 161
Scott, D. L., and Morse, H. D. Tuberculosis of kidney, 79
Seiler, Laura. Treatment of tuberculosis of eye, 80
Selective collapse, 114
Sensitization of cattle by avian tubercle bacillus, 9
Sensitizing guinea pigs to BCG, 104
Septicaemia, Tuberculous, in dog, 83
Serbst, C. A. See Leon, J. L., *et al.*, 117
Sergent. Lung abscess, 125
Settling characteristics of dusts, 161
Shadows, Perihilar, 58
Sharpe, C. T. Cyst of lung, 150
Sheldon, J. See Fineberg, S. M., *et al.*, 158
Silicosis, 22
——, Acute, 160
—— and asbestosis, 25
—— —— tuberculosis, 111
——, Experimental, and pneumonia, 26
—— in slate quarriers, 25
——, Prevention of, 25
Silico-tuberculosis, 27
—— in Cuba, 63
Simonet, Mlle. See Nègre, L., *et al.*, 93

Simpson, W. L., and Hughes, J. G. Bronchiectasis in tuberculosis of infancy, 53
Sindel, E. A. Empyema, 65
Singer, J. J. Bronchogenic carcinoma, 145
Sirois, A. Silicosis and asbestosis, 25
Sisti, M. Intracavitary drainage, 122
Skin reactions in guinea pigs, 85
—— tests with extracts from leprous spleens, 163
Skunk, Aspergillosis in a, 17
Slate quarriers, Silicosis in, 25
Smetana, H. F., and Kneeland, Y., Jr. Atypical bronchopneumonia, 137
Smith, C. E. Erythema nodosum in coccidioidomycosis, 39
Sokoloff, M. J. Lower lobe tuberculosis, 55
Solomides, J. Influence of peanut oil on tubercle bacilli, 94
Solomon, S. Friedländer lung infections, 130
Souto Granja, R. Tuberculosis of lung and larynx, 58
Spencer, J., and Warren, S. Radiation reaction in lung, 156
Spinal fluid, Meningococci and tubercle bacilli in, 81
—— fluids, Tubercle bacilli from, 100
—— fusion, 73
Spine, Tuberculosis of, 73, 74, 75, 76
Spirometry in collapse therapy, 90
Spleen, Tuberculosis of, 69
Spleens, leprous, extracts from, Skin tests with, 163
Spontaneous pneumothorax, 36
Sprague, E. See Myers, J. A., *et al.*, 45
Sputum, Culturing tubercle bacilli from, 98
—— examination of each lung, 98
Stahel-Stehli, J. Cholesterol granulomatosis of the lung, 32
Status asthmaticus, 157
Stéfko, W. H. Cavities and collapse therapy, 112
Steiner, P. Cancer of lung, 17
Steinke, C. R. Penetrating wounds of thorax, 34
Stemmerman, M., and Tchertkoff, I. Pneumonolysis, 116
Stewart, H. J., and Bailey, R. L., Jr. Pneumothorax and circulation, 91
Stomach, Tuberculosis of, 80
Strapping of chest, Oedema of legs following, 153
Street, D. See Sweet, P., *et al.*, 76
Streukens, T. L. See Myers, J. A., *et al.*, 47
Struthers, R. R. Sulfapyridine in pneumonia, 139
Students, Tuberculin survey of, 46
Study, X-ray, of apical tuberculosis, 108
Subphrenic origin, Exudates of, 141
Subpleural scars, Cancer of lung and, 20
Suction drainage of cavities, 3, 4, 5, 6, 7
Sulfapyridine in pneumonia, 139
Suppuration, Nonputrid, of lung, 125
——, renal, Pulmonary complications of, 141
Suppurative and neoplastic disease, Bronchography in, 140
Survey, Tuberculin, 52
——, ——, of students, 46
——, X-ray, in Negroes, 50
Surveys, Annual, 46
——, X-ray, Group, 49
Sussman, M. L. Nonputrid suppuration of lung, 125
Sutton, F. C., Britton, J. A., and Carr, J. G. Cardiopulmonary capacity, 91
Sweany, H. C. Tuberculosis of spleen, 69
Sweeney, J. A. Heart in tuberculosis, 63
Sweet, P., Bennett, G., and Street, D. Tuberculosis of spine, 76
Swine, Acid-fast bacterium in, 10
—— tonsils, Tubercle bacilli in, 10
Syndrome, Löffler's, 132
Syphilis and tuberculosis, 62
—— of lung, 129

Tabanera, J. A. Examination of faeces in tuberculosis, 98
Takano, M. See Yanagisawa, K, *et al.*, 83
T'ang, B. H. Y., and Hsu, C.-L. Transverse interlobar fissure, 58
Tchertkoff, I., and Stemmerman, M. Pneumonolysis, 116
Tchertkoff, J. C., and Reisner, D. Cystic bronchiectasis, 127
Temporary paralysis of intercostal nerves, 2
Test, patch, Tuberculin, 106
Tests, Skin, with extracts from leprous spleens, 163
Tetany, Respiratory, 31
Therapy, collapse, Cavities and, 112

Therapy, collapse, Spirometry in, 90
——, pollen, Oral, 158
——, Roentgen, for bronchogenic carcinoma, 147
Thoma, H. See Jarisch, A., *et al.*, 37
Thompson, B. C. Lymph node tuberculosis, 68
Thoracoplasty, Cavities after, 117
——, Intracavitary drainage after, 124
——, Perforation of pleura in, 118
——, Results of, 117
Thorax in track stars, 161
——, Penetrating wounds of, 34
Thorn, G. W., Howard, R. P., and Dayman, H. Electrolyte changes in tuberculosis of lung, 92
Thorner, Juliet. Erythema nodosum in coccidioides, 40
Thrombosis of superior vena cava, 40
Tice, F. Miniature films, 107
Tomography, 109
—— in tumors of lung, 147
Tompkins, E., and Douglas, B. H. Silico-tuberculosis, 27
Tonsillectomy and tuberculosis, 54
Tonsils, swine, Tubercle bacilli in, 10
——, Tuberculosis of, 69
Torek, F. Temporary paralysis of intercostal nerves, 2
Tosetti, R. Atherosclerosis of pulmonary artery and pulmonary emphysema, 29
Tracheobronchial tuberculosis, obstructive, Massive fibrosis in, 60
Tracheobronchiectasis, 127
Tracheobronchitis, Tuberculous, 67
Track stars, Thorax in, 161
Transient pulmonary opacities, 133
Transitory lung infiltrations, 11
Transverse interlobar fissure, 58
Trauma, Chylothorax following, 159
Treatment of empyema, 2
—— —— lupus vulgaris, 81
—— —— pneumonia, 139
—— —— tuberculosis of eye, 80
—— —— —— —— lung, 113
Trochanter, Tuberculosis of, 77
Trostler, I. S., and McNally, W. D. Pneumonoconiosis from Fuller's earth, 160
Tubercle bacilli, Anthracite and, 96
—— ——, bovine, Isolation of, 100
Tubercle bacilli, Culturing, from sputum, 98
—— —— from spinal fluids, 100
—— ——, growth of, Inhibition of, 97
—— —— in laryngeal content, 98
—— —— —— rats, 84
—— —— —— spinal fluid, Meningococci and, 81
—— —— —— swine tonsils, 10
—— —— —— urine, 99
—— ——, Influence of peanut oil on, 94
—— ——, Lymphocytes and, 84
—— —— on nonprotein medium, 93
—— ——, Virulence of, 93
—— bacillus, avian, Sensitization of cattle by, 9
—— ——, Morphological variation of, 95
Tubercles, miliary, Healed, 82
Tuberculin, Avian, 9
—— patch test, 106
—— reaction and Bang's disease, 11
—— survey, 52
—— —— of students, 46
Tuberculo-allergy, Histaminase and, 83
Tuberculoma of lung, 59
Tuberculosis and asthma, 60
—— —— industrialization, 45
——, apical, X-ray study of, 108
——, Asthma and, 61
——, Bone and joint, 71
——, ——, Early diagnosis of, 9
——, Brucellosis and, 34
——, Childhood, 53
——, ——, of lungs, 52
——, Conjugal, 47
——, Conjunctival, 80
—— control, 50
——, —— of, in India, 51
——, Cultural diagnosis of, 97
——, Cystic, of bones, 72
——, Decline of, 43
——, Diabetes and, 60
——, Diagnosis of, 108
——, Employee with, 46
——, Epidemiology of, 45
——, Examination of faeces in, 98
——, experimental, Oxidizing compounds and, 83
——, Extrapulmonary, 64
——, Follow-up in, 112

Tuberculosis, Heart in, 63
—— in adolescent females, 48
—— —— adolescents, 48
—— —— army, 49
—— —— children, 52
—— —— Chinese children, 53
—— —— nurses, 47
——, Leucocyte count in, 86
——, Lower lobe, 55
——, Lymph node, 68
——, miliary, Chronic, 59
—— of bladder, 79
—— —— bones, 71
—— —— endometrium, 78
—— —— eye, Treatment of, 80
—— —— infancy, Bronchectasis in, 53
—— —— kidney, 79
—— —— knee, 77
—— —— larynx, 67
—— —— lung, 58
—— —— —— and intestine, 58
—— —— —— —— larynx, 58
—— —— ——, Electrolyte changes in, 92
—— —— —— in Chinese, 51
—— —— ——, Treatment of, 113
—— —— spine, 73, 74, 75, 76
—— —— spleen, 69
—— —— stomach, 80
—— —— the bones and lung, 69
—— —— tonsils, 69
—— —— trochanter, 77
——, Pelvic, 78
——, Pregnancy and, 61
——, —— in, 62
——, Primary, in adults, 54, 55
—— program, 51
——, Respiratory function in, 89
——, Silicosis and, 111
——, Syphilis and, 62
——, Tonsillectomy and, 54
——, tracheobronchial, obstructive, Massive fibrosis in, 60
——, Veterinary Committee on, 8
Tuberculous adenopathy, 68
—— arthritis, 73
—— cavities, Bacteria in, 8
—— empyema, 66
—— endometritis, 78
—— epididymitis, 78
—— households, children of, Mortality in, 54
Tuberculous septicaemia in dog, 83
—— tracheobronchitis, 67
Tucker, W. B. See Bloch, R. G., *et al.*, 50
Tularemia of lung, 131
Tulsky, A. S. See Lackner, J. E., *et al.*, 78
Tumors, lung, Influenza and, in mice, 142
——, Malignant, of lung, 142
—— of lung, Tomography in, 147
Turley, L. A., and Dougherty, T. F. Lymphocytes and tubercle bacilli, 84

Ulrich, K., and Kartagener, M. Tracheobronchiectasis, 127
Ultraviolet radiation, 112
Unexpandable lung in empyema, 154
Ureterostomy, 79
Urine, Tubercle bacilli in, 99
Urquijo, C. A., and Saenz, A. BCG in vaseline oil, 85

Vaccarezza, R. F., Enquin, B., and Rodriguez, E. J. Tuberculin survey of students, 46
——, ——. ——., Erdstein, S. F., and Rey, J. C. Leucocyte count in tuberculosis, 86
Vaccination, BCG, 105
—— of guinea pigs with vole bacillus, 103
Vaccines, Cold, 142
van Wijk, A. M., and Patterson, H. S. Dust particles removed by breathing, 27
Variation, Morphological, of tubercle bacillus, 95
Vaseline oil, BCG in, 85
Velocity of pulmonary circulation, 92
Vena cava, superior, Thrombosis of, 40
Vera, H. D., and Rettger, L. F. Morphological variation of tubercle bacillus, 95
Verghese, M. C., and Benjamin, P. V. Diabetes and tuberculosis, 60
Veterinary Committee on tuberculosis, 8
v. Hayek, H. Histology of bronchi and bronchioles, 82
Virulence of tubercle bacilli, 93
Viswanathan, R. Lung abscess, 12
Vivoli, D. Bronchogenic carcinoma, 144
——, ——., and Bertelli, J. A. Tuberculosis of tonsils, 69
Vole bacillus, Vaccination of guinea pigs with, 103

Vorwald, A. J., Delahant, A. B., and Dworski, M. Experimental silicosis and pneumonia, 26

Waldbott, G. L., and Ascher, M. S. Respiratory allergy, 158
Walheim, T. Tuberculosis of spine, 73
Walter, Maria. Spirometry in collapse therapy, 90
Wang, S. H. See Hsieh, C. K., *et al.*, 146
Warren, S., and Spencer, J. Radiation reaction in lung, 156
Wartman, W. B., and Ingraham, E. S. Chemotropism, 96
Wassersug, J. D. Tuberculosis of trochanter, 77
Weber, H. Pneumonolysis, 116
——, ——. Suction drainage of cavities, 7
Weisman, S. A. Thorax in track stars, 161
Wells, A. Q., and Brooke, W. S. Vaccination of guinea pigs with vole bacillus, 103
Westdahl, P. R. Pulmonary embolism, 151
Westermark, N. Childhood tuberculosis of lungs, 52
Westwater, J. O. Antigenic depot excised, 105
Widmann, B. P., Ostrum, H. W., and Fetter, J. S. Tuberculous adenopathy, 68
Wight, A. E. Veterinary Committee on tuberculosis, 8
Wilkinson, M. C. Extrapulmonary tuberculosis, 64
Williams, W. C. Tuberculosis program, 51
Wise, N. B., and Poston, M. A. Hodgkin's disease and Brucella infection, 162
Wisnicky, W. See Hastings, E. G., *et al.*, 11
Wolff, B. P. Interstitial emphysema, 30
Wolpaw, S. E., and Hauser, H. Excavated bronchogenic carcinoma, 146
Woo, Theresa T. Tuberculosis in Chinese children, 53
Work, J. L., and Reichle, H. S. Healed miliary tubercles, 82
Wounds, Penetrating, of thorax, 34

X-ray damage to lung, 31
—— films, Miniature, 107
—— study of apical tuberculosis, 108
—— survey in Negroes, 50
—— surveys, Group, 49

Yanagisawa, K., Oobayashi, Y., and Takano, M. Oxidizing compounds and experimental tuberculosis, 83

Zaeper, G. Regulation of respiration, 86
Zeller, M. See Fineberg, S. M., *et al.*, 158
Zeyland, J., and Pisaecka-Zeyland, J. BCG vaccination, 105
Zorzoli, G., and Fojanini, G. Bacteria in tuberculous cavities, 8

# *WANTED*

To cooperate with institutions which seek to complete their sets and to replace files destroyed in the European War, the American Review of Tuberculosis is interested in obtaining the following back issues:

All numbers from March 1917 to February 1922 inclusive

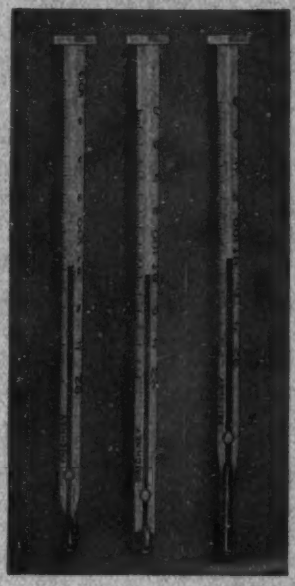
**1924—September only**
**1929—October only**
**1940—January to June**

If you have any or all of these issues in good condition and care to dispose of them, please communicate promptly.

*AMERICAN REVIEW OF TUBERCULOSIS*

*1790 Broadway, New York, N. Y.*





## "Tempglass Special" THERMOMETERS *for* Sanatoria Use

Especially adapted to sanatorium service... **Tempglass Special** thermometers have these important advantages.

- **Easier to Read**—94° to 106° scale calibrations are spaced 25% farther apart than on regular clinical thermometers.
- **"Shake" Tested**—Every Tempglass Special is selected for sanatorium use ONLY after repeated tests for easy shaking.
- **"Retreat" Tested**—Every instrument is carefully tested and checked against "retreating" . . . then "seasoned" from 4 to 6 months, and again inspected for retreating.
- **8 Certification Tests**—After repeated inspections and extra seasoning, each instrument receives eight certification tests for accuracy.
- **Economical to Use**—Our special tempering process makes the glass tough and durable. *Tempglass Specials* last longer . . . cost less, in the long run, than ordinary thermometers.

**Prices:** **Tempglass Special, any style bulb, per doz. . . . $6.50 per gross . . . $72.00**

Once tried, you will always want to use these better sanatorium thermometers. Order some today.

**FAICHNEY INSTRUMENT CORP.**
WATERTOWN, NEW YORK

***Faichney Thermometers Since 1888***

# There is a Council-Accepted high potency fish liver oil available that is advertised only to the medical profession and not exploited to the laity... It is called

## OLEUM PERCOMORPHUM

*(Liquid and Capsules)*

## Specify MEAD'S

*Yours for Keeping the Faith*

**MEAD JOHNSON & COMPANY**

EVANSVILLE, INDIANA, U.S.A.

Please enclose professional card when requesting samples of Mead Johnson products to cooperate in preventing their reaching unauthorized persons.